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THE DISEASES

OF

THE HEART AND THE AORTA.

THE DISEASES

OF

THE HEART AND THE AORTA.

AM S. YOUNG, PRINTER.

587
1855

TO

STAFF-SURGEON JOHN COLLIS CARTER, M.D.,

I DEDICATE THIS VOLUME,

WITH

TRUE RESPECT,

AND

IN REMEMBRANCE OF OUR LONG, UNFAILING,
AND MUTUAL AFFECTION.

TO

ROBERT WILLIAM SMITH, M.D.,

PROFESSOR OF SURGERY IN THE UNIVERSITY OF DUBLIN, ETC.

SIR,

In the composition of this work, while contending with difficulties inseparable from an attempt to combine the results of many years of labour, I have always been consoled by the thought that in dedicating it to you, I should be enabled to bear testimony not alone to the value of your contributions to Medical Science, but also to the signal benefits which your teaching and example have conferred upon the School of Surgery in this country.

WILLIAM STOKES.

PREFACE.

I DESIRE to state in brief terms the objects and nature of the following Treatise, which else, while we possess such works as those of Hope, Williams, Latham, and Walshe, might appear on the one hand uncalled for, and on the other insufficient. It seeks to embody the results of my clinical observations, continued almost unremittingly for upwards of a quarter of a century. Yet it is not to be taken as a record of every observation on Diseases of the Heart which may have been made by me during that time, but rather as expressing the state of opinion produced in my own mind by a long experience, even though I cannot recall many of the facts on which that opinion is founded. A work of this kind, if its author has had a sufficient experience, and especially if he has not sought to gratify his self-love by the advocacy of any new or peculiar doctrine,—than which there is nothing more likely to warp the judgment,—must always have a certain value. It is an attempt to convey to others the state of his own mind, the conclusions which he thinks may be safely arrived at, and the doubts and difficulties which he has been unable to solve or to remove.

I have sought to give to this work an essentially practical character; and at the cost of omitting much of what is new and interesting, I have made use of pathological anatomy, and the physical diagnosis founded upon it, only so far as these subjects bear on the every-day practice of our profession. The work, then, is not intended as a full treatise on Cardiac Pathology, nor yet on Physical Diagnosis, but it aims at the rational application of these branches of knowledge to Practical Medicine. Such a book ought to be useful, and is perhaps required; and whether the present attempt be successful or the contrary my readers may be assured that few of them will be more convinced of its imperfections than I am myself.

Without seeking to undervalue even in the slightest degree the many admirable works on Cardiac Pathology and Diagnosis which have been produced in our time, we cannot but admit that their effect on the mind of the inexperienced man is often different from that intended by their authors. His deficiency in clinical knowledge makes him overlook the great fact of the frequent complication of disease. He applies to a complicated case rules of diagnosis in which the isolation of disease is assumed; and while their apparent simplicity makes him confident in his powers, their inapplicability to the case in question leads him into grievous error. The diagnosis of the combinations of diseases even in so small an organ as the heart is still to be worked out; and until this be done, the rules of physical diagnosis founded on the presumed isolation of disease must be used with great caution. I cannot, even at the risk of being charged with understating the position of physical investigation at the present day, avoid expressing my opinion that a too great positiveness marks some of the statements in our standard works, and that the difficulties of special diagnosis are still infinitely greater than many might be led to believe.

In these remarks I do not wish to be considered as undervaluing the labours of the present advanced school of Physical Diagnosis. I believe that the object to which its members are directing their researches is the right one, and that the application of every true principle of diagnosis to practice will yet be discovered. I only wish, that, until the laws of this department of Vital Physics are fully and rigorously determined, no hasty or enthusiastic anticipations should be received as positive acquisitions to science; and as I wish this work to be considered as a treatise on a certain portion of the Practice of Medicine, and have kept this object steadily before me, I have made use only of such principles of diagnosis as may be safely received, and have avoided discussions on what still remains doubtful. I desire also to enter a protest against the tendency, still too prevalent in many schools, which would base the diagnosis of disease in great part, if not entirely, on the consideration of purely physical signs, to the exclusion of that important class of phenomena, which, for want of a better name, we are obliged still to call Vital. For there is nothing more calculated than this to cause the neglect of that first and greatest lesson in Medicine, which, while inculcating modesty and caution in diagnosis, makes us bring every possible light to bear on the case before us.

As the student, fresh from the schools, and proud of his supposed superiority in the refinements of diagnosis, advances into the stern realities of practice, he will be taught greater modesty and a more wholesome caution; he will find, especially in chronic disease, that important changes may exist without corresponding physical signs, —that as disease advances, its original special evidences may disappear,—that the signs of a recent and trivial affection at one portion of the heart may altogether obscure or prevent those of a disease longer in standing and much more important,—that functional alteration may not only cause the signs of organic lesion to vary infinitely, but even to wholly disappear,—that the signs on which he has formed his opinion to-day may be wanting to-morrow,—and lastly, that to settle the simple question between the existence of functional and that of organic disease will occasionally baffle the powers of even the most enlightened and experienced physician.

Yet, even since the times of Corvisart and Laennec there has been a great advance in our knowledge of cardiac and arterial disease: and, in proof of this assertion, it is only necessary to refer to the many works of a formal nature, and again to the numerous and equally valuable class of monographs which have been produced on these subjects during the last few years.

From unavoidable circumstances, the composition and printing of this work have been spread over a period of several years. This, I trust, will be taken as an apology for my omitting to notice some important additions to our knowledge, which appeared subsequently to the printing of the chapters devoted to the consideration of these subjects.

Thus I have been unable to notice the observations of Virchow on obstruction of the arteries in cases of disease of the aortic valve, nor the confirmatory researches of Dr. Kirkes, which have been recently published, nor, again, those of Dr. M'Dowel on the diagnosis of dilatation of the heart. The memoir of Dr. Gairdner, with reference to the relation between simple dilatation of the heart and the atrophic diseases of the lung, contains much matter for consideration, and should his views as to the influence of the dilating power of the thorax prove correct, they will throw a new light on diseases of the heart and also of the aorta.[1]

I am indebted to Dr. William Moore, of this city, not only for his kind and constant assistance during the progress of this work, but for the translation of that portion of the treatise of Skoda which contains the views of that author on the sounds of the heart, and for the copious Index with which this volume has been furnished. I wish, further, to express my grateful acknowledgments to Professor Smith and to Dr. Lyons, of this city, for their valuable aid.

[1] It will be observed that in the measurements of the heart, given in the note at page 273, the left ventricle is represented as being longer in women than in men, while all the other cardiac dimensions are greater in the male than in the female. This is clearly to be attributed to an error in the original, as at the five other ages at which Bizot gives the lengths of the left ventricle, the reverse appears to be the case; and in the paragraph immediately following the Table he observes, "that the dimensions of the ventricles in particular, as well as the general dimensions of the heart, are less in the woman than in the man."—*Mémoires de la Société Médicale d'Observation de Paris.* Tome Premier, 1836. Pp. 282, 283.

CONTENTS.

CHAPTER I.

INFLAMMATION OF THE HEART AND ITS MEMBRANES, ... 17

CHAPTER II.

DISEASES OF THE VALVES OF THE HEART, ... 144

CHAPTER III.

DISEASES OF THE MUSCULAR STRUCTURES OF THE HEART, ... 271

CHAPTER IV.

WEAKNESS OR DEFICIENT MUSCULAR POWER OF THE HEART, ... 314

CHAPTER V.

FATTY DEGENERATION OF THE HEART, ... 318

CHAPTER VI.

TREATMENT OF THE ORGANIC DISEASES OF THE HEART, ... 357

CHAPTER VII.

ON THE CONDITION OF THE HEART IN TYPHUS FEVER, ... 382

CHAPTER VIII.

DISPLACEMENT OF THE HEART, ... 468

CHAPTER IX.

RUPTURE OF THE HEART, ... 481

CHAPTER X.

DERANGED ACTION OF THE HEART, ... 497

CHAPTER XI.

ANEURISM OF THE THORACIC AORTA, ... 553

CHAPTER XII.

ANEURISM OF THE ABDOMINAL AORTA, ... 626

TABLE OF CASES, ... 667

INDEX, ... 675

A TREATISE,

ETC. ETC.

PART I.

DISEASES OF THE HEART AND AORTA.

CHAPTER I.

INFLAMMATION OF THE HEART AND ITS MEMBRANES.

Of the three forms of this disease described by authors, namely, endocarditis, myocarditis, and pericarditis, the last demands our especial attention, from its greater frequency, and from the marked character of its signs. Inflammation of the muscular portions of the heart, occurring independently of a corresponding state of either or both of its investing membranes, must be a very rare affection; and we are still but imperfectly acquainted with the history and symptoms of endocarditis. In most of the severe cases of carditis, the three great structures of the heart are probably engaged; and even though the muscular tissue may not exhibit the evidences of organic change, yet the signs of its irritative excitement and subsequent paralysis are plainly to be recognised. In truth, if we except the pain which so commonly attends serous inflammations, the remaining symptoms of pericarditis are to be referred less to the pericardium than to the muscular fibre.

It is true that the endocardium frequently participates in the disease, although pending the violence of the attack, the evidences of this lesion may be obscure or wanting. It does not appear possible to determine the presence or absence of endocarditis in the earlier periods of acute pericardial inflammation. The cardiac excitement can be otherwise explained, and even if the occurrence of valvular murmurs were diagnostic, their existence would be difficult or impossible to detect, from their being masked by the more prominent phenomena of acute pericarditis.

It is also true that in many cases of pericarditis a murmur is detected when the disease has been subdued, and all pressing danger removed; and this murmur may be permanent, and continue for months or years, till the patient die with the symptoms of valvular disease. Here we must believe that an inflammation of a valve has set in, either simultaneously with, or immediately subsequent to, the attack of pericarditis; and the frequency of this occurrence has led to the opinion, not only that pericarditis is commonly combined with endocarditis, but that many cases of the valvular diseases of the heart arise from inflammation of the endocardium. Yet we must be cautious in admitting these conclusions to their full extent. It may occasionally be found that the murmur, after existing for a period more or less extended, disappears, leaving the sounds of the heart in their natural condition, and the patient remains free from symptoms of valvular disease. The mere occurrence of murmur, even though immediately consequent on pericarditis, is not necessarily indicative of progressive valvular disease.

We are still unable to explain this occurrence satisfactorily. Is the murmur produced by a passing endocarditis which is not followed by organic change or deposit? Is it induced by atony of some portion of the muscular fibre, or may the cardiac orifices be altered by irregular or tonic spasm of the heart? This much is certain, that the occurrence of murmur following pericarditis should not necessarily lead to the diagnosis of valvular disease, in the ordinary acceptation of the term.

On the other hand, it too often happens that a violent attack of pericarditis may, under proper treatment, be subdued, and then the patient, having lost all symptoms of the malady, is considered as cured, and allowed to return to his usual habits. But in a short time a bellows murmur is established, which remains, with but little variation, for a long period, when the signs and symptoms of organic disease become manifest. This murmur is generally single, and accompanies the first sound, while the second remains unaffected. It obviously arises from disease of the endocardium, in all probability inflammatory, which has either co-existed with the pericarditis or has set in immediately subsequent to it. To the watchful physician there cannot be a time more full of anxiety than that immediately following the apparently successful treatment of an attack of acute pericarditis. Should his patient recover without the development of murmur, all is well; but the occurrence of this sign, and its permanency, are calculated to depress and discourage him in the greatest degree.

PERICARDITIS.

The earlier descriptions of this disease give but an erroneous idea of the affection, principally from this circumstance, that its more violent forms alone have been described. More recent investigations, however, show that the disease may occur in many gradations of intensity, and that it is frequently met with in such a mitigated form as really to present no symptoms by which it might even be suspected; in fact, in a form where its existence is only discoverable by physical examination. The idea of pericarditis is connected, in most men's minds, with severe and manifest symptoms, such as pain, tumultuous and irregular action of the heart, special modifications of the pulse, syncope, and so on, and it consequently happens that the disease is often overlooked. In some instances this is of little importance, as the processes of inflammation, exudation, and adhesion, go on to a favourable termination, without any medical interference, and the patient recovers from pericarditis, his physician being ignorant that he ever had any such affection. But in other cases the neglected inflammation, at first mild and unimportant, suddenly assumes a more virulent character, and the symptoms of pericarditis are developed when it is too late to overcome them by treatment.

In a practical point of view we may divide the cases of pericarditis into three classes. In the first are to be placed those in which there is but a slight, though general effusion of coagulable lymph. In the second we have superadded, the secretion of serum in abundance, causing distention of the sac. And in the third class we find, in addition to the preceding conditions, the signs of muscular excitement, if not of myocarditis.

Let us contrast these forms.

FIRST FORM.	SECOND FORM.	THIRD FORM.
Absence of pain or local suffering frequent. No sign of muscular excitement, nor any special character of pulse. No increase of dulness over the heart.	The local and general symptoms more decided, though often very trifling. Irregular action of the heart and pulse, often more manifest in the advanced periods. Remarkable increase of dulness over the heart.	Local distress, often extreme even at the outset. Tumultuous action of the heart. Irregularity of pulse. Dyspnœa, orthopnœa, œdematous swellings, syncope, death.

These forms are not merely different in the degree of violence of the disease, but draw their distinctive characters from other circumstances. That there is a progressive increase in the violence of the original inflammation, as we ascend from the first to the third form, may be admitted. The great characteristic of the second form, however, is the effusion of fluid, while that of the third is the irritative or inflammatory excitement of the muscles of the heart. It is this which causes the great suffering, and, as we shall presently see, constitutes the danger in the advanced stages of the disease; for there can be little doubt that death occurs by syncope, induced by paralysis of the left ventricle, the result of its preceding excitement or inflammation. The muscles of the heart are then in the same condition as that of the intercostals after violent pleuritis; and when the weakened organ has not only to propel the column of blood, but to struggle with the pressure of a large body of fluid, while its action is clogged by a deposit of coagulable lymph, it is no wonder that it should fail to fulfil its function.

In explaining the mode of death in pericarditis, however, too much importance has been attached to the effect of pressure by the superincumbent fluid. It is singular how much pressure the heart is capable of bearing without any important disturbance of its functions. Thus in dislocations to the right side from an empyema of the left pleura, though the pressure exercised must be much greater than that in an ordinary case of pericardial effusion, the action of the heart is rarely disturbed. I have published a case in which pericarditis attacked a heart thus displaced, yet without any injury or disturbance of the action of the organ; and Mr. Adams has observed a case of long-continued pressure of the heart, so great as to fold up part of one ventricle, in which the heart endured this effect for a considerable length of time.

If we again refer to the analogous case of the intercostal muscles and diaphragm in pleurisy, we find that these muscles are capable of resisting an amount of pressure greater than that which occurs in most cases of pericardial effusion. Distention of the side, dislocation of the heart, and of the lung, may be observed before any yielding of the muscular portions of the chest; so that the conclusion is forced upon us, that, so long as the contractility of the fibre is not weakened by disease, all these muscles are capable of bearing a great increase of pressure without their functions being suspended.

Two conditions of the muscles may be supposed to exist. One, simple atony or paralysis; the other a true myocarditis, attended with deposition of new matter among the fibres, or by ulcerative absorption. In the first of these conditions recovery is possible, just as we see in pleuritis that the action of the paralyzed intercostals is restored, while in the second the organ appears to be irreparably injured.

We may then conclude, that when death takes place as a consequence of pericarditis, the contractile power of the left ventricle at least has been seriously injured, and that the organ is either simply paralyzed, or that its structure has been altered more or less deeply by inflammation of the fibres themselves.

When we examine the pathology of myocarditis we shall return to this subject.

But it must not be forgotten that in many cases of severe pericarditis there is complication with other diseases, local or general, and that we may be in error in attributing death to the cardiac inflammation alone. The patient may die with a severe pericarditis, but not necessarily from the effects of the local disease, simply considered. That such was the nature of many of the severe cases given by Louis appears certain. In his first case the disease affected not only all the structures of the heart, but also the lungs, stomach, and hepatic portion of the peritoneum. In another case the affection was evidently connected with intermittent fever and nervous disease. In a third case the pericarditis was complicated with delirium tremens, which had been improperly treated, and extensive gastro-pulmonary disease.

Of this combination I have seen several examples, in which the pericarditis, though intense, was but one of a group of irritations, all of them secondary to, or at least complicated with that form of typhus or typhoid fever which follows on an excessive debauch and exposure to cold, and which sets in and is accompanied with delirium tremens.

In this terrible disease, we may sometimes find a true typhus fever, with characteristic petechiæ, while in other cases the fever is of a typhoid type, in connexion with a group of local inflammations. This disease is generally fatal. I have found cerebritis, bronchitis, gastro-enteritis, double pneumonia, and pleurisy, coexisting with the pericarditis in these cases.

We may divide the cases of pericarditis into the uncomplicated and complicated forms. Under the first head, however, we include those cases in which the muscular structure and the endocardium may be engaged.

Uncomplicated.

a. Inflammation of the serous membrane alone.
b. Inflammation of the pericardium with combination of endocarditis and possibly of myocarditis.

Complicated.—Under the head of complicated pericarditis we may make two great divisions:

a. Complication with general disease.
b. Complication with one or more local diseases of structures unconnected with the heart.

Under the first of these heads may be arranged the following cases:—

a. Combination with rheumatic fever.
b. Gout.
c. Phlebitis.
d. Typhus fever.
e. Dropsy.
f. Delirium tremens.
g. Intermittent fever.

Under the second we may enumerate a great number of examples, most of which must be familiar to the clinical observer.

- *a.* Pericarditis associated with pleuritis, which is generally of the left lung.
- *b.* Combined with pleuro-pneumonia of one or both lungs.
- *c.* Associated with a group of typhoid inflammations.
- *d.* Superadded to chronic hypertrophy of the heart.
- *e.* Acute pericarditis supervening on a chronic empyema.
- *f.* In connexion with fatty degeneration of the heart.
- *g.* Induced by ulcerative perforation of the pericardium.

To this list many other examples of the association of pericarditis with diseases of various organs might be added.

On taking a review of the symptoms of pericarditis, we find that, as the disease may occur under a great variety of circumstances, its symptoms present a singular want of constancy in character. The disease may be absolutely latent, so far as symptoms are concerned, or be indicated by signs of extreme cardiac and general suffering. The picture of the affection, as given in the older nosological works, only belongs to the more violent forms, and is imperfect even with respect to them. But while the symptoms are so varied, the physical signs are constant, and of easy interpretation, and the same principles of diagnosis apply to every form of the disease. And it must be admitted that, of all the thoracic diseases, there is none of which the diagnosis so much depends on physical investigation. Hence, as the signs are so well marked, their study will give us a more comprehensive view of the various stages of the affection than we could get by examining the symptoms in the first instance. We shall then examine some cases illustrative of the different forms of the disease, and its combinations, and so be enabled to study its general history with reference to vital symptoms.

Let us then examine the physical signs in this affection.

Up to the year 1833, when the signs of pericarditis were more carefully studied, the diagnosis rested mainly on negative evidence, that is to say, that in a case of manifest inflammation within the thorax, if we could satisfy ourselves that the disease was neither pleuritis nor pleuro-pneumonia, we might, with great probability of being right, make the diagnosis of pericarditis.

In the year 1824, in a work by Dr. Collin, we have the first notice of the physical signs of pericarditis. The following are his observations on this subject:

"We have only once observed the sound analogous to the creaking of new leather. It occurred in a patient who died of chronic pericarditis. This sound continued for the first six days of the disease, but disappeared as soon as the local symptoms indicated a slight liquid effusion into the pericardium. M. Dervilliers, intern pupil at the Hospital of St. Antoine, observed it at the same time in a patient whose symptoms indicated pericarditis. He was not aware that the phenomenon had been already observed in this disease, and did not avail himself of it in his diagnosis. In this case it is to be regretted that no dissection was recorded. On another occasion M. Dervilliers examined the body of a man who had presented this phenomenon during the whole of his stay in hospital. A chronic pericarditis, producing thick, false membrane, and numerous vegetations over the heart, was discovered; the number of adhesions was small, and the pericardium did not contain a single drop of serosity. Perhaps this sound would be a constant symptom of pericarditis before the occurrence of liquid effusion, fugacious in cases where the disease runs its course in a short time, but of longer duration in chronic cases."[1]

Collin referred the friction sound, as observed by him, to a dry state of the serous membrane, the first effect of its inflammation, and compared it to the sound produced in certain cases in the knee, when we produce a friction between the patella and the condyles of the femur. There are, however, strong grounds for believing that the friction sounds in pericarditis indicate that lymph has already been effused. From the rarity of death in the very first stages of the disease, it becomes difficult to declare that a merely dry state of the membrane will not suffice to produce the sign, and there seems no reason why it should not do so. On the other hand, it is certain that in all the cases in which a double friction sound was observed, and in which there was a dissection, lymph was found covering the pericardium. The researches of Dr. Mayne show that in cases where the symptoms and subsequent phenomena concurred in proving the existence of an incipient pericarditis, some time elapsed before the friction sound was developed. I have myself verified this observation of Dr. Mayne's.

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[1] Les diverses Methodes d'Exploration de la Poitrine.

It is admitted that in the natural state of serous membranes, the gliding of one surface on the other, so as to produce the least possible amount of friction, is admirably provided for by the exquisite smoothness of the surfaces, which are further bedewed with a lubricating exhalation. Should the surface, under the influence of inflammation, become merely dry, it is almost certain that some friction phenomena would be developed, particularly in the pericardium, where the membrane is pressed upon by the comparatively firm and unyielding mass of the heart. But this state cannot continue long, and, though unable to point out the exact time when the friction sounds from mere dryness pass into those produced by a roughened state of the surface, we need not regret the difficulty, as it must have relation to but a short portion of time, and does not bear on any practical question.

In the roughened state of serous membranes from inflammation and exudation of lymph, two classes of phenomena are produced:

First. Sounds having a generic character, yet varying according to the different physical conditions of the parts. They have been termed the friction sounds.

Second. Phenomena discoverable by the touch. For example, when the hand is applied over the region of the inflamed organ, sensations as of two surfaces rubbing and grating one on the other, are often perceptible. These signs are of more rare occurrence than the former, and are often absent when the sounds are manifest. They imply that the lymph is in a state of unusual consistence or hardness, and probably also that the surface is but little bedewed with serosity. And hence, as might be expected, they are generally better developed during the earlier periods of the disease than when, after the absorption of the serous part of the effusion, and under the process of cure, the surfaces again come into contact.

Among the conditions which favour the production of friction signs perceptible by the hand, the resisting nature of the organ covered by the inflamed membrane occupies a prominent place; and it is probable that the greater frequency of these signs in pericarditis, rather than in pleuritis, is referable to the unyielding nature of the structure of the heart, as compared with that of the lung. Whoever has once grasped the living heart of an animal, can understand what a hard and solid mass it presents during the systole. We further find, that, in the case of peritoneal friction, the sign has been principally observed where the inflamed membrane invests some organic tumour or solid viscus. Can we then explain the rarity of the tactile friction signs in the advanced and resolutive stages of pericarditis, by supposing a weakened state of the heart, which interferes with the vigour of its contractions, and renders it, during the systole, less hard and resisting?

Third. Signs discoverable by percussion. In many cases of simple pericarditis, where the heart has not been previously diseased, the sound on percussion over the organ remains unaffected; but when the pericardium is distended by solid, fluid, and gaseous secretions, modifications of the sound, with reference to its character and to the extent of dulness, are always produced.

GENERAL ADHESION OF THE PERICARDIUM.

The occurrence of obliteration of the sac of the pericardium has been enumerated among the causes of some organic diseases of the heart, especially of its hypertrophied and dilated conditions. It is supposed that, from the difficulty experienced by the heart in contracting under this condition, the muscles increase in strength and volume, until a true hypertrophy is induced. This doctrine, so far as it relates to the production of hypertrophy in consequence of adhesion of the pericardium, must not be admitted in its full extent, notwithstanding that it has been strongly advocated by Dr. Hope. "I have never," he says, "examined, after death, a case of complete adhesion of the pericardium, without finding enlargement of the heart, generally hypertrophy with dilatation.

This sufficiently demonstrates the tendency of the affection."[1] In another place he observes: "How adhesion occasions hypertrophy is easily understood, for, first, inflammation is probably a cause of hypertrophy, and secondly, the organ must increase its contractile energy in order to contend against the obstacle which the adhesion, by checking its movements, presents to the due discharge of its functions, and, as explained in the article on hypertrophy, increased action leads to increase of nutrition.

"The cause of the co-existent dilatation is not less manifest. As the shackled organ transmits its contents with difficulty, it is constantly in a state of greater congestion than is natural, and, as is more fully explained in the article on dilatation, permanent distention is the most effective cause of this affection. When the muscular substance has been softened by the previous inflammation, as frequently happens, dilatation takes place much more readily, in consequence of the deficient elasticity or tone of the heart's parietes."[2]

Without denying that a general adhesion may induce hypertrophy and dilatation, experience leads me to doubt that such an effect necessarily or even commonly follows the condition indicated. I have often found the heart in a perfectly natural condition, with the exception of an obliterated pericardium. It was neither hypertrophied nor atrophied, and the patient had exhibited no symptoms of heart disease for many years before death. In one case seven years had elapsed between the death of the patient from hepatic disease, and the attack of pericarditis which obliterated the sac. During this period no symptoms of disease of the heart were manifested. Again, if we take the cases of simple pericarditis with recovery, we cannot doubt that adhesion more or less complete has occurred; and yet any increased liability of such patients to enlargements of the heart has not come under our observation. It is in those cases of pericarditis which we have before indicated, and where valvular disease is either co-existent with or subsequent to the first inflammation of the sac, that hypertrophy and dilatation appear as remote consequences of pericarditis. In the cases of recovery without murmur, we have little apprehension of the after-occurrence of organic disease.

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[1] Last edition, p. 181.

[2] Ibid. p. 182.

It has been stated to me by Professor Smith, that he has found general adhesion of the pericardium coinciding with atrophy or with hypertrophy of the heart, in a nearly equal frequency. In some of the cases of atrophy the change was simple, consisting essentially in a diminished volume, with perhaps a paler colour, of the heart, while in others a true fatty degeneration had commenced. In another series the heart showed the fatty degeneration invading, more or less completely, the entire of the cardiac walls. And it is a remarkable fact, recorded by the same observer, that he has always found ossification of the pericardium, which we may hold as the extreme of the obliterating process, attended with atrophy of the heart.

The application, then, of the doctrine that muscle increases in volume and force in proportion to the resistance to its action, must be received in a qualified manner when we apply it to the elucidation of diseases of the heart. It is true that we often see hypertrophy of that cavity of the heart which has to propel blood through a diminished valvular orifice; but we may fairly draw a line between the cases of obstruction to muscular action from obliterated pericardium and valvular disease. In one, as in adhesion, the normal condition of the muscle is interfered with, and so the contraction diminished; while in the other the muscle, being free to act, increases in power, just as the voluntary muscles do when trained by exercise.

Analogy seems to favour views contrary to those of Dr. Hope. Obliteration of the pleura is commonly followed by a diminished volume of the lung. In chronic peritonitis with general adhesion, the intestinal tube is more frequently found thinned, contracted, and weakened, than in the opposite condition. And were we to extend our examination to the case of the voluntary muscles, it would not be difficult to demonstrate that the existence of a mechanical obstacle to their free contraction is followed by atrophy.

On the whole we may conclude,—

First. That obliteration of the pericardium does not necessarily induce any manifest change in the condition of the heart.

Second. That, where alteration of the muscular condition of the heart is found in connexion with this obliteration, it is not necessarily a state of hypertrophy, but is often one of an opposite nature.

Third. That the cases of valvular obstruction and of adhesion of the pericardium are not parallel, inasmuch as that in one case the heart is free to act, while in the other its motions are prevented or interfered with.

Fourth. That obliteration of other serous membranes is more often followed by atrophy than by hypertrophy of the subjacent organs.

Fifth. That atrophy of the voluntary muscles is the ordinary effect of whatever interferes with their free action.

There is a case, however, which, in this inquiry must not be passed by without notice, namely, the existence of a true muscular aneurism of the ventricle, co-existing with an adhesion which corresponds to the tumour or sac. It is difficult to say whether this adhesion is the cause or consequence of the aneurism. Yet, if we adopt the first opinion, it still does not go far in strengthening the views of Dr. Hope, for we can easily understand that, while the rest of the heart remains free to act, the adherent portion will be first impeded, then paralyzed, and finally yield, so as to allow of a local accumulation of blood. Here it is partial adhesion which causes dilatation, and we cannot infer from this that a general adhesion would induce hypertrophy.

If, however, we adopt the opinion of Rokitanski, that partial aneurism of the heart arises from an inflammatory action originating in the endocardium or in the muscle, we can comprehend how a partial adhesion would be produced, and stand then as a consequence and not a cause of the disease.[1]

PHYSICAL DIAGNOSIS.

It is plain, that as the physical diagnosis of pericarditis depends on the existence of some of the products of inflammatory secretion within the sac, we cannot directly apply it to the very first stage of the disease. In this respect, however, pericarditis forms no exception, as in all other diseases of the chest a mechanical alteration of some kind must exist before physical signs are produced. The first stage, then, of pericarditis, or that anterior to any change of the surface of the sac, is undiscoverable by physical means. But it does not follow that the use of auscultation is of no avail even under these circumstances, for we may often be led to a suspicion of pericarditis by finding that there are no physical signs of inflammation of the lung or endocardium.

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[1] See also Hasse's book, translated by Dr. Swaine. Sydenham Society, 1845, p. 141.

How long this state of pericarditis may last it is difficult or impossible to state, but the period is generally so short, that the detection of the disease on its entry into the second stage, or that in which it affords physical signs, is sufficiently early for all practical purposes.

We owe to Dr. Mayne an important series of observations of pericarditis, in some of which the patient was under observation for a certain time preceding the appearance of physical signs. In the first case it was not until the third day after symptoms of pericarditis had set in, that physical signs were discovered, although on each day the stethoscope was carefully employed. The symptoms were great epigastric tenderness, particularly severe when pressure was directed towards the pericardium; an extremely distressing sense of weight about the heart, the impulse of which was very strong, but regular; the pulse 130, small, wiry, and regular. The patient was treated for acute pericarditis, yet the friction sounds did not appear until the third day of the disease, so that the pericarditis must have existed certainly for twenty-four, and probably for thirty-six hours, before physical signs were produced. In another case the same period seems to have elapsed between the invasion of the disease and the appearance of the friction signs. The impulse of the heart was very great, contrasting remarkably with the pulse at the wrist, which was rapid and small; the sounds of the heart were rapid, but unaccompanied by friction, and the impulse communicated a considerable shock to the ear; friction signs were not observed until the third day of the pericarditis.

It is possible that the period anterior to the formation of lymph in these cases might have been forty-eight hours, but it is very probable that it was much less. Dr. Mayne concludes that in the present state of our knowledge there is no stethoscopic sign which can be considered pathognomonic of the first stage, which, he says, is the more to be regretted, as this is, of all others, the period at which most benefit might be expected from active antiphlogistic treatment. It has been already observed, however, that pericarditis, as to its want of physical signs in the first stage, forms no exception in the class of thoracic diseases; it would be well, indeed, if every acute disease could be positively ascertained within thirty-six hours of its invasion; and it is possible, too, that the omission of that active antiphlogistic treatment, still so often employed in the first stages of inflammation, might be of no great detriment to the patient.

This practical lesson, however, is derivable from what has been said, namely, that in a case of suspected pericarditis in its early periods, the absence of friction signs must not lead us to conclude that the pericardium is safe. I have known several days to elapse before the appearance of friction signs, in a case where pericarditis was superadded to inflammation of the left pleura.

Finally. If the disease be of a violent and dangerous character, we shall almost certainly have *symptoms* of a special nature to guide us, before the appearance of the friction signs. And on the other hand, if the case is a mild, dry pericarditis, there is no great chance of injury to the patient from its being overlooked for one or even two days.

The physical signs of pericarditis may be classified as follows:—

First. Sensations of friction communicated to the hand. To these we may give the general term of tactile signs.

Second. Friction sounds; the "*attrition murmurs*" of Hope.

Third. Extension of dulness over the heart, resulting from liquid effusion.

Fourth. Friction signs attended with or preceded by valvular murmurs.

Fifth. Signs of eccentric pressure analogous to those of empyema.

Sixth. Signs of excitement of the heart.

Seventh. Signs of weakness or paralysis of the heart.

It may be stated generally that the tactile and acoustic signs vary according to the following circumstances:—

1. The state of the effused lymph.
2. Its extent.
3. The existence or non-existence of fluid.

4. The advance or arrest of the process of organization.
5. The process of obliteration of the cavity.
6. The repetitions of inflammation.

I have already indicated these conditions in my original memoir; the following, however, must be added:—

7. The existence of air in the pericardial sac.
8. The distention of the stomach with air.
9. The combination with pleuritis of the left lung.
10. The force and volume of the heart.
11. The combination with recent or previously existing disease of the valves.

There is no serous inflammation which presents such a difference in the physical constitution of its products as pericarditis, and hence the friction phenomena in this disease are more singular and varied than in peritonitis or pleuritis; and they further present more remarkable changes in short spaces of time. The products of inflammation present every form of effused lymph. It may be as hard as cartilage, or form a soft, diffluent coating or net-work over the heart; again, serous or bloody fluid, in various quantities, may be also effused; or the heart may be found bathed in a homogeneous purulent liquid, or with its surface completely studded over with minute warty masses, so as to resemble the coarsest rasp.[1]

With the exception of the leather creak sound of Collin, and some of the loudest rasping sounds, the friction phenomena are, in general, singularly localized, and are not heard beyond the actual region of the heart. In many instances we find that on removing the stethoscope but a single inch from the spot where the sound is loud, it totally ceases, although we still hear the ordinary sounds of cardiac pulsation.[2]

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[1] The production of an extremely indurated false membrane, as the result of acute disease, is of importance, as we generally attribute induration to chronic disease. I have communicated to the Pathological Society some examples of acute induration of the lung, where the organ presented the hardness of chronic pneumonia. Dr. Corrigan, also, has recorded similar facts.

[2] Dr. Hope, when referring to this observation, says, that he suspects that the limitation of the murmurs results from nothing more than their weakness, aided, perhaps, in some cases, by their being generated on the posterior surface of the heart; "for," he observes, "when a murmur generated on the anterior surface is

As might be expected, we find the most intense friction sounds under two conditions: one a great degree of induration of the lymph, and the other, the dry state of the surface. Under these circumstances, the rasping and rubbing sounds are sometimes produced with extraordinary intensity, and it often happens, at least in the earlier stages of the case, that the friction sensation is communicated to the hand.

In other cases, however, the sounds, though distinct, do not convey the idea of so unequal and resisting a surface, but resemble the rubbing together of two sheets of paper or parchment. In such cases the lymph will probably be found of a soft consistence. And there is a third class of cases in which the friction sounds convey the idea of the rubbing together of two surfaces but little roughened, and bedewed with a liquid secretion. The sounds in such cases are sometimes so soft, equable, and gentle, as to render it necessary that the patient should hold his breath for a few seconds in order that we may fully observe them.

It is not, however, to be believed that each of these modifications marks a separate case. In some instances of dry pericarditis, the characters of the sounds undergo but little change, if we except a gradual diminution of intensity; but in other cases the signs are presented in every possible variety of character.

The extent of the effused lymph materially affects the friction signs. In most cases, at least before any process of adhesion has commenced, the lymph is spread over the whole surface, and we then observe the signs of friction with the systole and diastole of the heart over the entire cardiac region. But friction signs confined to one portion of the heart are commonly observed, and we may divide such cases into two classes.

First. Cases where the signs are discoverable, in the earlier periods, over but a limited portion of the heart. In this condition they may continue, the disease appearing to remain singularly

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loud, I see no reason why it should not be extensively propagated."—*Op. cit.* On this I have only to remark, that in my original memoir I merely stated the fact of the limited transmission of these sounds, unless under certain circumstances; and I have given a case in which the sounds were extensively heard. I must add, that in many cases where the localization of the signs was observed, the anterior as well as the posterior surface of the heart was completely roughened by exudation.

localized up to its termination in cure. We generally observe these signs corresponding to the sides of the ventricles, rather than to the apex or base of the heart.

Second. Cases in which, after the general extension of the friction signs over the heart, adhesion takes place at the apex and lateral portion of the ventricles; under which circumstances the friction sounds become localized, and often remain at the base of the heart for a considerable period of time.

Although the modification of the friction sounds principally indicated by Collin was the *leather creak*, "*bruit de cuir neuf*," yet this appears to be the rarest of the forms met with. We are yet ignorant of the exact nature of the conditions requisite for its production, and can only say that it indicates dry pericarditis. But we know that other forms of the friction sound are much more frequent.

Two circumstances having the effect of modifying these sounds must be here mentioned. One is the application of local antiphlogistic means; and the other, the employment of pressure over the heart.

Nothing, indeed, can be more remarkable than the rapidity with which these signs are altered by the application of leeches over the heart, by a blister, or a poultice. They change within a few hours, even from the loudest rasp, with distinct vibration to the hand, into a soft murmur, while the tactile signs disappear. By this means we are sometimes, in cases of doubt, enabled easily to distinguish between the pericardial and valvular sounds. I do not believe that, to the well-educated ear, the difficulty of distinguishing these phenomena is as great as some writers have supposed. Yet cases occur which, when seen for the first time, may cause doubt. Thus the occurrence of a local pericarditis in a case of pre-existing organic disease of the heart, is a combination that may be difficult to determine. I lately saw a case of this kind where the patient had long laboured under symptoms of disease of the heart, probably the fatty degeneration. I found over the right ventricle a rasping sound, and as I could not ascertain whether this was a new or long-existing sign, I did feel difficulty in diagnosis. The sign, however, as I was informed by the attending physician, disappeared in a few days, after the application of a blister and the use of a few mercurial pills.

I have spoken of the effect of pressure. If, while the stethoscope is applied, we make a strong downward pressure with the hand, or increase the pressure of the head on the ear-piece, we shall often find a notable increase in the loudness and distinctness of the friction sounds; so that, in a case passing towards cure, we may reproduce, to a certain degree, the harshness and loudness which existed in the earlier periods of the attack. The same effect can be even better produced by causing an assistant to make pressure with the open hand over the cardiac region, during the application of the stethoscope. As might be expected, this modification by pressure varies directly as the elasticity of the chest. It is very remarkable in children, in women, and in young, feeble men.[1]

This mode of proceeding may be adopted in certain cases where we are in doubt as to the nature of the sounds. I have not made any extensive series of observations on the effect of pressure in modifying the character of valvular murmurs, but it is certain that the pericardial sounds are much more influenced by pressure than those arising from valvular disease.

The complication with a liquid effusion modifies all the phenomena of pericarditis. It may cause their suspension after the disease has for some time existed in the dry state, while in the resolutive stages of the case, its absorption is followed by their return. It is also attended with changes in the sound of percussion over the heart, the extent of dulness furnishing a measure of the effusion. In fact, the phenomena of pleurisy with effusion, and of pericarditis, are mutually illustrative; and as in pleurisy it may happen that, from the simultaneous effusion of lymph and fluid at the commencement of the disease, we may get the signs of liquid, without preceding friction phenomena, so in pericarditis the sac may be distended without our having ever observed the friction signs. But this occurs much more frequently in inflammation of the pleura than in that of the pericardium.

Again, as in pleuritis the existence of a liquid effusion does not necessarily prevent the occurrence of friction signs, so in pericarditis does the same rule apply. Of course, in both cases the co-existence of friction sounds and extensive dulness is rare, but of the fact there is no doubt, and I have ascertained that this curious combination is much more frequently met with in pericarditis than in pleuritis. I have often found the friction sounds to remain at the base of the heart, long after extensive liquid effusion had taken place into the sac; and it is particularly necessary to insist on this, as it has been stated by some writers, that the third stage of pericarditis is not accompanied by *frottement.*[1]

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[1] This proceeding may be objected to by some, as productive of distress to the patient; but in most cases, unless in the earliest and most acute stages, pressure on the pericardium does not cause any great inconvenience or suffering.

In a case observed in the Meath Hospital some years ago, in which there was extensive dulness, the friction signs could be heard when the patient lay on his back, but disappeared on his assuming the erect position. The explanation of this is obvious. A case is given by Dr. Corrigan,[2] in which the pericardium was enormously distended, so as to reach to the first rib. When the patient sat up, the friction sounds diminished, and sometimes altogether disappeared, but became well marked whenever he lay on his back. The heart was covered with a pulpy lymph, and there was a vast effusion of liquid into the sac.

Having thus taken a general view of the direct signs of pericarditis, and the ordinary sources of their modification, let us, before alluding to some of the rarer phenomena, examine the succession of physical signs in the two principal forms of the disease, namely, the dry pericarditis, and that which, at some period, is attended with liquid effusion.

CASE I. *Simple Dry Pericarditis.*—Development of friction sounds and tactile vibrations. The sounds may at first be general or partial, and then spread over the whole surface of the heart. They may be at first soft, but rise to a maximum of roughness and loudness; when they commence to decline, becoming softer and more feeble. This change generally takes place first towards the apex, and extends to the base of the heart. They finally cease, the cardiac region remaining all the time with its natural sound on percussion.

CASE II. *Pericarditis with Liquid Effusion.*—Friction signs are first developed with various degrees of intensity, but are generally less loud and rough in this case than in the preceding one. They soon disappear, either wholly or over a great extent, being still heard in some cases, principally at the base of the heart. The dulness diminishes, and with the return of clearness the friction signs re-appear, though still generally feebler than in their first stage; then finally subside, leaving the sounds of the heart natural. The tactile signs may or may not be present at the commencement or resolution of the disease, but are seldom so well developed as in dry pericarditis.

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[1] Dublin Journal of Medical Science, First Series, vol. vii. p. 278.

[2] See Transactions of the Pathological Society of Dublin, December, 1842.

It is plain that in both these cases the diagnosis of an adhesion of the pericardium, more or less complete, can be easily made, not, however, from any direct signs of the condition itself, but from the fact of our having observed the exudation of lymph, with or without liquid, formed in a serous sac, and passing into organization. I more than doubt that there is any certain physical sign of adhesion of the pericardium, and have never been able to verify the sign relied on by Dr. Hope of the double jogging impulse. It appears more than probable that, out of the great number of cases observed in the Meath Hospital, where the numerous changes of the friction signs were accurately investigated, many of them resulted in adhesion rather than in resolution; yet in none was the sign in question developed after convalescence. Indeed, from our general knowledge of the history of serous inflammations, we must conclude that resolution without adhesion must be of very rare occurrence in pericarditis; and consequently it is fair to infer that in most of the cured cases of the disease an adhesion has really taken place.

We may now consider the remaining causes of modification of the friction signs.

I. *Co-existence of Air with the usual Products of Inflammation.*

There seems no reason to believe, that if air be occasionally produced in the pleura or peritoneum, when in a state of irritation, that the same should not occur in the pericardium. On this subject I have no anatomical evidence to produce, but I feel satisfied that in one case at least I observed the phenomena of pericarditis with pneumatosis. The patient was a young man of lymphatic temperament, who had laboured under an attack of acute pericarditis for a few days before I saw him. On my first examination he presented the usual signs of dry pericarditis, with a considerable effusion of lymph of the ordinary consistence. The rubbing sounds, though loud and distinct, had nothing unusual in their character, and the patient suffered but little distress. After two or three days I saw him again, and found that his state had become very much altered. His appearance was haggard and worn, and he complained of extreme exhaustion, which he attributed to a total deprivation of sleep. This was induced by the extraordinary loudness and singular character of the sounds proceeding from the cardiac region; for though up to this period the rubbing sounds were distinctly perceptible by means of the stethoscope, the patient was quite unconscious of their existence. They had suddenly, however, become so loud and singular, that the patient and his wife, who occupied the same apartment, were unable to obtain a moment's repose. On examination, a series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sounds of indurated lymph, or the leather creak of Collin, nor those proceeding from pericarditic with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met so extraordinary a combination of sounds. The stomach was not distended by air, and the lung and pleura were unaffected, but the region of the heart gave a tympanitic *bruit de pot felè* on percussion; and I could form no conclusion but that the pericardium contained air in addition to an effusion of serum and coagulable lymph.

In the course of about three days the signs of effusion of air disappeared, leaving the phenomena as they were at the first period of the case. The convalescence of this patient was slow, and the rubbing sounds continued for an unusual length of time.[1] His recovery was ultimately perfect.

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[1] There is a circumstance connected with this case worthy of being recorded as illustrative of the influence of the depressing emotions in retarding the processes of cure in disease. After the disappearance of the signs of air, I was in hopes that

This case I believe to have been one of pure pneumo-pericarditis. We have as yet no information as to this combination. If we refer to Laennec we find the observation that the temporary existence of air in the pericardium causes a great degree of loudness of the heart's sounds; but he does not speak of the effusion of air in connexion with actual pericarditis. Dr. Hope doubts whether, in Laennec's cases, the air was in the pericardium, and suggests that the loudness of sounds was caused by the distention of the stomach. It is remarkable, however, that in the case now recorded we had both the symptoms and signs of pericarditis; and in addition to the clearest evidence of air in the pericardium, there was this remarkable circumstance, that the sounds of the heart were audible at a great distance from the patient.

Dr. Graves has observed a case of pneumo-pericarditis from fistulous opening into the sac, which is of great value, as determining the character of the physical signs in this combination.

A woman aged 25 was attacked with acute hepatitis, which ended in abscess. In a few days *the hepatic tumour emitted a tympanitic resonance.* On the twelfth day from this occurrence she was attacked with pains in the cardiac region, followed by violent beating of the heart, and a sensation of burning heat below the left breast. On the next day she presented friction sounds of various kinds over the heart, and these were soon complicated with a new set of phenomena. Immediately under the mamma a peculiar metallic click was occasionally heard, giving the idea of a fluid dropping in the pericardium. This sound ceased when pressure was made over the heart. On the third day from the invasion of the

the patient would be speedily restored to health; but day after day elapsed, and no progress seemed to be made in the organizing process. The rubbing sound remained unchanged, notwithstanding the employment of all the means I could devise to bring the case to a successful issue. I observed that the patient was depressed and melancholy, and on inquiring from his wife whether he had any mental suffering, I was told that he had had great fears as to his spiritual state, and was full of doubts on many points of his religious belief. Under these circumstances I asked a clergyman distinguished for his talent and eloquence to visit my patient. This interview was followed by the best results. Next day the rubbing sounds had become softer; the visit was repeated, and on the third day all morbid signs had disappeared. That the process of organization in this case was prevented or delayed by the depressed condition of the patient's mind, there can be no doubt. The recovery of the patient was complete.

pericarditis, rubbing sensation was communicated to the hand, and the sounds assumed the character of an emphysematous crackling, obscuring both sounds of the heart. This was most distinct along the middle and inferior parts of the sternum, but could also be heard to the left of the mamma. The metallic click became more audible, but was not produced in a regular way. On the day before death, a loud metallic ticking, audible at each stroke of the heart, could be heard combined with the emphysematous crackling and the other sounds. A slight bellows murmur existed at the region of the left nipple.

It was found that the sac of the hepatic abscess had formed two openings—one near to the pyloric orifice, communicating with the stomach, and the other passing directly through the union of the diaphragm and pericardium into the sac. This perforation was large enough to admit the middle finger. The pericardium was intensely inflamed, and covered with great quantities of lymph in various degrees of consistence.

This most important, and, as far as I know, unique case, shows us an example of pneumo-pericarditis by fistulous opening, and may be compared with the ordinary case of pneumothorax by perforation of the pulmonary pleura. Here the supply of air was manifestly from the stomach, taking a course through the hepatic abscess in the first instance by the original perforation, and from thence passing into the pericardium.[1]

If the preceding cases are compared, any doubt that could be entertained as to the real nature of the first of them must be removed; for in both the physical signs were closely similar, and they only differ by the addition of the signs of perforation in the example described by Dr. Graves.

The following case of perforation of the sac, producing pneumo-pericarditis, must be studied in connexion with that which has now been given. For the particulars of this case I am indebted to Dr. B. M'Dowel. The *post mortem* appearances were exhibited at the Pathological Society of Dublin.

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[1] I have greatly abridged this case from Dr. Graves' Clinical Medicine. It may be placed in that important category of cases, which, independent of their rarity, may be taken as introductory to the diagnosis of new forms or combinations of diseases, or of affections previously known, but for the discerning of which no clear rules existed. It is to the diagnosis of pneumo-pericarditis by perforation, what Dr. Beatty's case of abdominal aneurism, and Dr. Adams' of fatty heart, are to the diagnosis of the respective diseases of which they furnish examples. See Dublin Hospital Reports, vols. iv. and v.

A policeman, aged 29, of robust frame, was admitted into the Whitworth Hospital in July, 1846, complaining of cough and other anomalous symptoms. He stated that a month before his admission to hospital he had exposed himself to cold by taking off his coat whilst in a profuse perspiration. This was followed in the course of three or four days by a severe stitch, low down in his right side; for this he had himself bled, and experienced relief from the operation. In a few days, however, pain of the same kind returned, but now it was confined to his left side. He had himself blooded a second time, but without experiencing any advantage. He had at this time also profuse perspirations, cough, and some pains in his chest, but no rigors. The matter expectorated was of a dark colour.

On this man's admission to hospital, which took place a month after the commencement of the above symptoms, no physical evidence of disease could be discovered in either side; but after some days he was attacked by a stitch in his right side, which was relieved by a blister; he very soon after, however, began to complain of pain in his left side; this soon became agonizing, and attended with severe dyspnœa. The day after, the following were the symptoms and signs observed:—The expectoration had become copious, purulent, and fetid; his breath was also extremely fetid. Dyspnœa, amounting to orthopnœa; voice faint, at times nearly extinct. Countenance haggard, pale, and anxious. Pulse 110, weak. Some cough. Delirium at night, and slight diarrhœa. The physical signs gave evidence of a large cavity, containing air and fluid, in the antero-inferior region of the left side of the chest; here was heard metallic tinkling, *bourdonnement amphorique*, and splashing of fluid, caused by the action of the heart; these sounds were produced by making the patient breathe deeply, and with them could be heard faintly the normal cardiac sounds, but no respiratory murmur. Percussion yielded a perfectly clear sound over these regions; but it was clearer than that yielded posteriorly over the corresponding part of the lung, although no part of this side was dull; the respiration in the upper part of the left lung was faint. Posteriorly from the centre downwards *frottement* was audible; and over the base of the same lung a coarse crepitus was heard. No local fremitus on either side, owing to weakness of voice. In the right lung a fine crepitus was audible over the base posteriorly. Anteriorly, and circumscribed to a limited space, about the eighth rib, below the mamma, was heard a whiffling sound, resembling cavernous respiration. The symptoms and signs of respiration, as described, continued, with the exception of dyspnœa, which was relieved by opiates:—He, however, became delirious on the 26th of July, and died during the night, six days from the supervention of the violent symptoms.

Dissection, twelve hours after death:—On opening the thorax, a greatly distended pericardium, concealing the left lung, was brought into view; and on cutting into it, evidences of intense inflammation were seen. The sac was greatly thickened, and lymph, rough like mortar, lined its opposed surfaces; it contained about six ounces of pus, having the consistence and colour of milk. A round fistula existed on the right wall of the sac, which led into a small anfractuous cavity, near the second fissure, in the upper lobe of the right lung; this contained matter similar to that found in the pericardium. The bases of both lungs were solidified from a double cause, first, from a deposit of miliary tubercle, and secondly, from pneumonia. Apices of both lungs healthy. Universal inflammation of the left pleura, with lymph spread over its surface, but there was no adhesion. On passing a current of air through the trachea, it was observed to rise through the fluid contained in the pericardial sac; the pericardium, when cut into, contained air.

Let us compare this case with that given by Dr. Graves. In both instances, a fistulous opening of the pericardium was followed by sudden and severe pericarditis, and by the effusion of air into the sac. In Dr. Graves' case, the signs, though singularly modified, were still those of pericarditis; while in that by Dr. M'Dowel these were wanting; and a group of signs, closely resembling those of the ordinary emphyema and pneumo-thorax, were produced. This is probably to be explained by the greater amount of the aeriform effusion, and by the character of the products of inflammation in this case. The heart was found bathed in a creamy, homogeneous, purulent fluid: and it is almost certain, that no friction sign ever was or could ever have been developed, as we may suppose, that from the moment of the perforation, the heart became enveloped by the contents of the abscess in the lung.[1] The greater amount of air, too, may be referred to the direct communication with the lung. In Dr. Graves' case, on the contrary, the air was derived from the stomach, and by a tortuous course passed into the pericardium.

It is to be noted in this case, that there was not only no augmentation of the sounds of the heart, but that they were rendered feeble. Was this produced by the intervention of the aeriform fluid, just as in pneumo-thorax the vesicular murmur becomes indistinct or inaudible, even before the lung has completely collapsed?

Thus it appears that two classes of metallic phenomena of the pericardium, very different in their cause and nature, may be met with. In one class the character is from the actual existence of air within the pericardium, while in the other it is caused by the distention of a neighbouring viscus with air.

II. *Distention of the Stomach with Air.*

The influence of flatulent distention of the stomach, and in some cases of the large intestine, in modifying the sounds on percussion in hepatization of the lung, particularly on the left side, has long been known. This condition often leads to errors. The same cause affects all signs derived from auscultation; and thus we find that the crepitating and mucous râles of bronchitis and pneumonia, the friction sounds of pleuritis, and, finally, the sounds of the heart and the friction signs of pericarditis, may present a distinct metallic character. I have observed this to affect every morbid sign in a case of double pleuro-pneumonia and dry pericarditis.

With respect to the latter affection, however, we merely find that the rubbing sounds are metallic, but there is none of the singular emphysematous crackling, the metallic click, or the loud gurgling and churning of air and fluid that have been observed in pneumo-pericarditis. As might be expected, too, this character is temporary, and irregularly intermittent, and I have succeeded in immediately removing it by the administration of a carminative draught or a turpentine enema, and restoring to the thoracic sounds their ordinary character.[1]

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[1] An important case is given by Dr. Mayne, in which about eight ounces of thin pus were found in the sac of the pericardium. There were no false membranes, and no form of friction sound was ever developed. I shall again allude to this case. See Dublin Journal of Medical Science, First Series, vol. vii., p. 274.

III. *Modifications of the Friction Sound from a Complication with Pleurisy of the Left Lung.*

Strictly speaking, the peculiarities thus produced have no reference to any change in the acoustic character of the signs of pericarditis, but arise from the production of similar sounds in the pleura, which, as they correspond with the motions of the lung, differ in rhythm from those of the pericardial disease.

It may be inquired, if we have such a condition of the pleura as will give the ascending and descending friction sounds, may we not also have a sound of friction produced merely by impulse of the heart against the pleura, thus causing three pleural friction sounds? It is further to be ascertained, that this sound, so unfrequent, may not occasionally be a double sound, for we know that the heart often gives a double impulse, in which case four pleural friction sounds might be produced. Lastly, it is possible, that if we had the combination of pleurisy with pericarditis, not less than six friction sounds might be developed. Of these two would be those of the ascent and descent of the pleura, two from the double impulse of the heart impinging on the pleura, and two from the friction produced within the pericardium itself.

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[1] Nothing can be more meager than the information given by writers on diseases of the heart on the subject of pneumo-pericarditis. Laennec says nothing as to its causes, except when it arises as a cadaveric condition, or occurs in the last periods of life. Dr. Hope doubts whether the cases indicated by Laennec were really examples of the disease in question. Louis himself does not describe pneumo-pericarditis, nor has he any case of this condition resulting from ulcerative perforation of the sac: it is not even mentioned by him. *Mem. sur la Pericardite.* And Rostan merely suggests that the sensation of fluctuation observed by Senac and Corvisart may have been caused by this complication. "I have sometimes," says Laennec, "been able to announce its presence, from the supervention of an increased resonance over the lower part of the sternum, and from the existence of the sound of fluctuation produced by the action of the heart, and by deep inspirations."—Forbes' Translation, chap. 24. The fact of our occasionally being able to hear the heart at a great distance is dwelt on by him as an indication of pneumo-pericardium; yet it is remarkable that this sign was not present in either Dr. Graves' or Dr. M'Dowel's cases. On the other hand, it existed in the case which I have recorded. It may be that in fistular pneumo-pericarditis the sounds of the heart are not augmented, from the want of that tension of the sac which we may presume to exist in effusions of air, without fistula. A case, too, might be anticipated, in which a valvular fistula of the pericardium might be attended with increased pressure of air within the sac. Bouillaud gives a case by M. Bricheteau, in which a sound similar to that of water agitated by a mill-wheel, was found in the pericardial region, and which evidently proceeded from the alternating motions of the heart. On dissection, an effusion, resulting from chronic pericarditis, was found. The purulent matter was extremely fetid, and when the sac was opened, a rush of gas escaped. In this case, also, percussion of the pericardium, practised before the sac was punctured, gave the "*bruit de flot.*"—*Traité des Maladies du Cœur*, 1836, p. 332.

It is now several years since a case occurred in the Meath Hospital, in which the sounds of the heart striking against the pleura occurred. The signs, in addition to those ordinarily observed in pleurisy, were the friction sounds of ascent and descent, and a friction sound, attended with vibration perceptible to the hand, and synchronous with the impulse of the heart, which continued when respiration was suspended. The sound ceased when the patient assumed the erect position. On dissection, a very small quantity of unorganized lymph was found at the posterior surface of the heart, but the pericardium presented none of the appearances usually observed in cases presenting distinct friction signs. The pleura, on the other hand, was covered with a copious exudation of lymph, which had become granular on its surface and semi-cartilaginous in structure. It is to be observed that, in this case, the heart was dislocated downwards from old emphysema.

There is yet another source of multiplication of the sounds in pericarditis, exclusive of any affection of the pleura. A condition of the heart may be observed, in which one of the sounds becomes, as it were, doubled. This may arise, in nervous cases, in carditis, and, as we shall hereafter see, in that condition of the heart where inflammation of the organ is threatened. It is rare, however, that its occurrence is found to modify the sounds in pericarditis; yet I have observed a case where there was no physical evidence whatever of pleurisy, yet in which the rhythm of the heart was triple, one friction sound coinciding with the single, and two with the double sound of the heart. The case was one of rheumatic fever of ten days' duration, and the friction sounds at first were feeble, and passing into a soft bellows murmur: general and local bleeding greatly reduced the heart's action, and then *the friction phenomena became more distinct.* In four days the friction sounds were triple, and in the recumbent position, accompanied with a metallic click, but this peculiarity ceased when the patient sat up. In two days more the triple character of the sounds disappeared, and in a short time all traces of pericarditis had vanished.

As to the causes of this doubling of one of the sounds of the heart, we can as yet offer no satisfactory explanation.

It is easy to comprehend, that, according to the relative rates of rapidity of the cardiac and pulmonary actions, which will of course vary in different cases, different rhythms or modes of succession of the sounds will be met with in different cases, or in the same case at different stages of its progress.

V. *Influence of the Force and Volume of the Heart.*

It will be unnecessary for us to dwell at any length on the last source of modification of the friction sounds, namely, the force and volume of the heart. In general, other things being equal, the loudness of the friction sounds will vary with the force of the heart; and we might imagine a case in which, notwithstanding the existence of a quantity of lymph on the heart, the sounds would be feeble or absent from the want of a sufficiently active muscular contraction.

I have had but little experience of the influence of the volume of the heart upon the sounds in pericarditis. I do not think that in the combination of enlarged heart with inflammation of its serous covering, there is any change produced in the nature of the sounds; but it seems probable, that the extent to which they may be heard is increased. I have already noticed the remarkable fact of the limitation of even the loud friction sounds to the cardiac region, as one of great value in diagnosis. Yet we are not to infer, that in cases of extension of the sounds, the heart is necessarily enlarged. The leather creak sound may be heard over the whole chest without any enlargement of the heart; and in a case which

I have already published, and in another recorded by Dr. Watson, the same result was found. The heart, in both cases, was thickly studded with granules of a semi-cartilaginous structure.

Dr. Graves has some good observations, however, on the increase of volume of the heart in causing extension of the friction sounds. He gives a case of the combination of hypertrophy and dilatation with pericarditis, in which the motions of the heart were accompanied by two loud, prolonged sounds of equal duration, but of different tones. The first was a *bruit de scie;* the second was a musical sound, closely resembling that made by rubbing the moistened finger on glass. These sounds were very distinct under both clavicles, but were not heard in the carotid or subclavian arteries. In the course of twenty-four hours the musical sound changed to a well-marked leather creak.

The heart was found hypertrophied and dilated, and coated with lymph, the most recent effusion of which appeared at its base; the valves, lining membrane, and blood vessels, were all healthy. A large quantity of fluid occupied both pleural cavities; a circumstance considered by Dr. Graves to have been an additional cause of the extension of the friction sounds, as it acted by pressing the heart against the walls of the chest. It is remarkable that the pulse was only 70 or 72.[1]

But, without denying that the existence of an enlargement of the heart may cause an extension of the friction sounds, I believe that this phenomenon will be found to depend more on the nature of the sounds themselves, than on the extent of the inflamed surface. We know that a great extension of sounds may occur without alteration in the volume of the heart, and it is remarkable that, in Dr. Graves' case, even the musical sound was inaudible at the apex of the organ.

On the whole, I incline to the opinion, that the mere enlargement of the heart only causes extension of these sounds, in virtue of the greater amount of surface engaged; so that under these circumstances, the sounds, were it not for other conditions, would not be audible beyond the actual region of the heart, although this region was morbidly enlarged. I have already published a case of a greatly enlarged heart, affected with pericarditis, in which, although repeated observations of the state of the lung were made, no friction sounds were ever detected, except over the region of the heart; and these were only discovered on the day before the patient's death. The case, too, was one peculiarly adapted for the extension of friction sounds, for the heart was not only greatly enlarged, but presented the appearances of an acute hemorrhagic pericarditis supervening on a chronic disease, as shown by an effusion of lymph of a soft consistence, and of the colour of blood, with, at the same time, vast depositions of a semi-cartilaginous hardness; the heart's action was strong, *and the friction vibration manifest*, a point of importance to be observed, as it might be supposed that the want of extension of friction sounds was caused by the overlaying of the indurated lymph with the more recent and softer effusion.

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[1] Clinical Medicine.

We have seen that in Dr. Graves' case there existed copious liquid effusions into both pleuræ, which he considers, by pressing the heart against the walls of the chest, assisted in the extension of the friction sounds. My experience, however, leads me to conclude, that the friction sounds are not necessarily extended, even though the heart be under extreme pressure. I shall presently adduce two cases of empyema, one of the right, the other of the left pleura, in which great eccentric displacement occurred. In the last case, indeed, the heart, at the time it became affected with pericarditis, was dislocated far to the right side, yet even under this amount of pressure the friction signs remained confined accurately to the heart in its new situation. In the case of empyema of the right side the pressure was so great as to depress and alter the form of the liver, and to cause dulness extending across the median line; in this case, too, the friction signs were completely localized.

Finally, we have never observed that, even when rendered more distinct by pressure with the hand, the friction sounds extended beyond their original situation.

Upon the whole, I incline to the opinion that extension of the sounds in pericarditis is to be referred to the special character of the sounds themselves rather than to any effect of internal pressure.

The last source of modification is the existence of valvular disease, either contemporaneous or previously existing. In certain cases this combination may cause some obscurity in diagnosis, but I believe that writers have over-estimated the amount of the difficulty. If we take the case of a previously existing valvular disease, the following circumstances will serve as a means of diagnosis:—

First. The actual acoustic character of the sound.

Second. Its arising from a point comparatively deep-seated, and where it is at its maximum.

Third. Its not being equably or nearly equably diffused over the surface of the heart.

Fourth. Its greater extension over the thorax.

Fifth. Its frequent want of the double character, the first or the second sound of the heart being often unattended with murmur.

Sixth. Its being frequently transmitted along the aorta and its primary branches.

Seventh. The absence of friction sensation communicated to the hand.

On the last character it is to be observed, that the valvular tremor, like the sound, has, in many cases, a point of greatest intensity, and is not extensively diffused, as in pericarditis. Indeed, unless in some of the rare cases of varicose aneurism, the maximum point of the tremor is generally determinable without difficulty.

There is, perhaps, a greater difficulty in settling the question when the disease affects the mitral valve, leaving the aortic orifice free; for in this case we have no transmission of the murmur along the vessels. A careful consideration, however, of all the phenomena, will, in almost every case of doubt, lead us to a correct conclusion.

I have already observed, that the signs of pericarditis must have often been mistaken for those of diseased valves. But their sudden supervention in a case where they had never before existed, the accompanying sign (when present) of the rubbing sensation communicated to the hand, the rapid change of situation, the equally rapid modification by treatment, and the occurrence of the signs with both sounds of the heart, in a case which previously presented no evidence of organic disease, form a combination of circumstances which can hardly mislead.

But when it happens that, coincident with the attack of pericarditis, a diseased action is set up in the valve, the determination of the latter may be difficult, during the continuance of the true friction murmurs. If the valvular sign be, as it commonly is, a bellows murmur, it may be completely masked by the loudness of the friction sounds, and only become manifest on their cessation. For some time, too, before these latter have wholly subsided, but when they have lost much of their loudness and roughness, it may be difficult to say how far the two sounds are intermingled. Yet the determination of the question is of importance only as relating to the prospects of the patient. It is a question of prognosis rather than of treatment; and the case in question illustrates this important maxim, that in acute affections, when the diagnosis of the diseases of adjacent parts is difficult or impossible, it is often unnecessary, so far as treatment is concerned.[1]

The development of valvular murmur, in recent cases of pericarditis, does not appear to me to possess the value assigned to it by Dr. Hope and Dr. Watson as an indirect sign of pericarditis. I have never observed the valvular to precede the friction murmur, though the signs are often found to co-exist;[2] and I believe that in these cases the diagnosis of endo-pericarditis may be made. Dr. Hope seems to have overrated the frequency of the combination, or, to speak more correctly, has underrated the occurrence of simple pericarditis, in which there is no valvular murmur developed, either during the acute stage of the disease or even after its cure by adhesion. On the other hand, that the cure of acute pericarditis is often unfortunately imperfect, inasmuch as the patient recovers with an established valvular murmur, is too true; and though years may elapse before the valvular disease produces its full effect in embarrassing the circulation, he has, from the time of his apparent recovery, a slowly advancing, insidious, and unconquerable disease.

We have, however, observed some cases in which a murmur with the first sound of the heart, though distinct for many days after recovery from pericarditis, gradually subsided and did not re-appear. Was this the result of retrocedence of valvular inflammation, or was the murmur one of those sometimes attendant on a weakened state of the heart? The latter supposition appears most probable.

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[1] In two of the cases recorded by Dr. Mayne, no murmur preceded the attrition sounds, although at the time of observation the pericardium was manifestly in a state of inflammation. Increased action of the heart was the principal sign. See Dublin Journal of Medical Science, First Series, vol. vii.

[2] Whether any effect of adhesion of the sac, by interfering with the free action of the muscles, might for a time cause murmur, is worthy of inquiry.

It may be inquired, whether any assistance can be derived, in the diagnosis of pericarditis, from studying the acoustic signs which are proper to the muscular contraction of the heart, simply considered. This is a subject on which new researches are required, yet I cannot but think that some important results would follow from the investigation. It is to be determined whether any sign, independent of the irregularity of the heart's action, could be discovered, which would indicate the extension of disease to the muscular structure; whether the ringing sound of the ventricular contractions may be taken as a proof of the first stages of myocarditis; whether any purely muscular murmurs are developed; and lastly, whether in the advanced stages of inflammation, the muscular sounds become weakened or destroyed.

With reference to the last point I can state, that I have observed the disappearance of the first sound of the heart in cases of severe pericarditis; so that if we except the irregularity of action, the signs closely resembled those of the softened or weakened heart in typhus fever; and although the cause of this condition is pathologically different, yet, physically considered, it is the same in both diseases, and proceeds from the weakened state of the muscular fibres, resulting in one from the effects of inflammation, in the other from relaxation, with or without the interstitial typhoid deposit.

Before noticing the signs derived from percussion, it will be convenient to state, in separate propositions, the conclusions derivable from what has been now advanced; and as it will not be without value to ascertain what progress has been made in the elucidation of the whole subject since the date of my memoir (1834,) I shall place first in order the eleven propositions which contained the result of my researches up to that period, and then continue the series, so as to embody whatever subsequent experience I may have had of the friction signs of pericarditis.

1. That in cases of pericarditis with effusion of lymph, the rubbing of the two roughened surfaces causes sounds perceptible to the ear, and vibrations communicable to the hand, by which the disease can be easily and surely recognised, even when all other indications are absent.

2. That the more rough the state of the serous membrane, the more distinct will these signs be.

3. That they accompany both sounds of the heart, but are most distinct with the first sound.

4. That they are in general audible only over the region of the heart.

5. That they present themselves with various modifications of character, but sometimes resemble the sounds produced by extensive valvular disease.

6. That they are most distinct when the region of the heart continues with its natural sound on percussion, but that the existence of fluid does not necessarily imply their complete subsidence.

7. That they may re-appear either after the absorption of fluid from the sac of the pericardium, or the supervention of new inflammation.

8. That the sounds may continue when the sensation of rubbing is no longer perceptible by the hand.

9. That they are singularly and rapidly modified by direct antiphlogistic treatment.

10. That by observing the progress and mutations of these signs, we can trace the process of organization or of obliteration of the pericardial cavity, judge of the effect of treatment, and accurately ascertain the state of the pericardium.

11. That, hence, it must be admitted, that auscultation is of direct utility in pericarditis, and that the diagnosis no longer rests on negative signs.[1]

12. That the vital symptoms of acute pericarditis, with the exception of pain, are to be referred more to irritation or excitement of the muscular portions of the heart, than to the corresponding states of its external or internal membrane.

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[1] See Dublin Journal of Medical Science, First Series, vol. iv. (1834.)

13. That acute pericarditis is often so latent as to be discoverable only by physical signs.

14. That this latent form, however, may suddenly assume a manifest and violent character.

15. That the cases of this disease may be divided into three great classes.

a. Simple dry pericarditis, with little or no muscular excitement.

b. Acute pericarditis with liquid effusion, and with, in many cases, a greater amount of muscular excitement.

c. Acute pericarditis with effusion, and with severe symptoms of muscular suffering, as indicated, first, by excitement, and secondly, by paralysis.

16. That death in pericarditis may be generally attributed to syncope or pseudo-apoplexy, caused by paralysis of the heart.

17. That the effect of the pressure of the effused fluid on the heart has been probably overrated.

18. That the weakness of the heart may proceed from simple atony or paralysis, or results from true myocarditis.

19. That in the more violent forms of pericarditis there is often a complication with other diseases, both local and general.

20. That the first stage of pericarditis may be observed without the existence of any friction sign.

21. That this stage is of short duration, so that the want of friction signs in the first stage cannot be adduced as an argument against the utility of physical signs in pericarditis.

22. That the length of this period probably varies from six to thirty-six hours.

23. That the absence of friction signs in the first stage is of less importance than appears at first sight; for if the disease be violent and dangerous, it is indicated by symptoms, and if it be mild and simple, its discovery in the very first stage is of comparatively little importance.

24. That the existence of air in the sac, whether originally secreted (pneumo-pericarditis) or introduced by a fistulous opening, modifies the friction sounds in a special manner, producing crackling, gurgling, and metallic sounds, sometimes audible at a

great distance from the patient. This is, so far, confirmatory of the suggestion of Laennec.

25. That in the first of these cases, on the absorption of the air, the ordinary character of the friction sounds may be produced.

26. That distention of the stomach with air may give a distinct metallic character to the friction sounds.

27. That the sounds most commonly heard over a large surface of the chest are the leather-creak sound of Collin, and the loud rasping sound proceeding from indurated lymph.

28. That lymph may be produced in the pericardium, of an almost cartilaginous hardness, as a result of acute disease.

29. That the extension of the sounds seems more related to their actual character than to the pressure exercised on the heart, or the volume of the organ.

30. That, nevertheless, pressure exercised on the cardiac region is often followed by an increase of the loudness of the friction sounds, and of the distinctness of the tactile signs.

31. That in cases of combination with pleurisy of the left lung, not less than five attrition sounds may be produced. Of these two are from the heart, two produced by the ascending and descending motions of the lung, and one from the impulse of the heart against the pleura.

32. That, consequently, a variety of rhythms of the friction sound may thus be developed.

33. That enlargement of the heart does not necessarily imply that the friction sounds will be heard beyond the space occupied by the organ.

34. That although in certain stages of some cases of pericarditis a difficulty may arise in determining the exact nature of the sounds, as distinguished from valvular murmurs, yet that this difficulty, which is only temporary, appears to have been overrated.

35. That we are to depend for accuracy in diagnosis on the actual acoustic character of the signs; on their diffusion or concentration at a point of greatest intensity; on their being superficial or deep-seated; on their amount of extension over the thorax; their double or single character; their transmission or non-transmission along the course of the vessels; on the presence and character of the tactile signs; on their constancy or variability in character and seat; and on the effect of treatment in their modification.

36. That the diagnosis of an adherent pericardium can only be made with certainty in cases where we have observed the phenomena of effusion and organization of lymph.

37. That adhesion may co-exist with atrophy as well as hypertrophy of the heart, and lastly, may be found with a heart unaltered in its capacity or muscular condition.[1]

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[1] See the works of Hope, Walshe, and Barthe and Roger, where the principles of the differential diagnosis are given. I announced most of these characters in my communications on Pericarditis, Dublin Journal of Medical Science, First Series, vols. iii. and iv. (1833–1834.)

I confess to a feeling of natural pride, when I find that my labours on the subject of the diagnosis of pericarditis have elicited the testimony and approval of such authorities as Dr. Forbes and Dr. Hope; and I think that in transferring to these pages the recorded sentiments of these observers, I may be fairly excused.

Dr. Forbes, after referring to the propositions at the conclusion of my paper, says:—"The facts so concisely announced in the preceding propositions are of such practical importance, that I must recommend the attentive consideration of every one of them to the reader. It is most gratifying to those who were the early and, by some, the suspected advocates of auscultation, to find it gradually working its way to the high places of the profession, and vindicating its true philosophical character by successive improvements and discoveries, among the most valuable of which I do not hesitate to regard those of Dr. Stokes, detailed in the present note." See the translation of the work of Laennec by Dr. Forbes, Art. Pericarditis.

In his classical work on Diseases of the Heart, Dr. Hope has the following remarks:—"The history of the discovery of the various murmurs of endo-pericarditis is as follows:—After the discovery of 'creaking of new leather' by Collin, in 1824, Dr. Latham, in 1826, discovered a bellows murmur with the first sound, as a sign of *rheumatic* pericarditis. He communicated this to me in the same year; and I found, and published in the first edition, in 1831, that the murmur accompanied not only rheumatic, but any kind of pericarditis; that it sometimes attended the second as well as the first sound; that it was referrible, not to the pericardium, but to co-existent endocarditis, and that it was the earliest and best sign of inflammation of the heart. Dr. Elliotson had, unknown to me, published in the previous year, that the murmur was referrible to endocarditis. I can now distinctly recollect various cases in which I noticed that the murmurs were 'creaking,' 'anomalous,' 'extraordinary;' and I entertain no doubt that these were attrition murmurs: I failed to discriminate them, because, during the last ten years, not having had a fatal case of acute pericarditis, I have not had the opportunity of post-mortem verification. Had Collin given a happier name than '*bruit de cuir neuf*' to attrition murmurs, I have no doubt that they would have much sooner been recognised. Though the honour of giving the first clue to this class of murmurs belongs to Collin, and, though Broussais, as will presently be shown, noticed the sound like rubbing of parchment, yet the merit of satisfactorily unravelling the whole subject is, in my opinion, to be awarded to Dr. Stokes,

SIGNS DERIVABLE FROM PERCUSSION.

We use percussion with advantage in every form and stage of pericarditis. Its results are negative or positive. Negative when, as in dry pericarditis, there is no alteration of the sound, and positive when the increase of liquid effusion extends the line of dulness, or when by absorption the natural sound of the heart is restored.

It has been supposed, that in carditis there is an extension of dulness, not to be attributed to liquid effusion, but to the inflammatory turgescence of the heart. Such an occurrence is, at least, doubtful, and we may safely assume, that the variations of sound in pericarditis depend on the actual amount of the effusion.

According to Hope, the presence of half a pint of fluid is sufficient to cause a perceptible increase in the line of natural dulness; and the same author has observed, that as compared with the dulness in hypertrophy, this dulness from effusion mounts higher up, in the direction of the great vessels.

The effusion causing this dulness being almost always inflammatory, it happens that friction signs precede, and up to a certain point co-exist with the extending dulness. They then commonly cease for a time, to reappear when, from absorption of the fluid, the inflamed surfaces come into apposition. But there are cases in which, though modified in intensity, the rubbing sounds continue through the whole period of effusion. They are comparatively feeble, and confined to the base of the heart, while the dulness is extended, but are developed over a large portion of the organ, when the liquid effusion is removed. At this latter period, the rubbing sensation communicated to the hand may or may not be present.

The dulness, so far as it extends, is complete, and we do not know any means by which, from its mere character, it can be dis-

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Dublin Journal of Medical Science, First Series, vol. iv., Sep. 1833.) Apparently, (without being aware of the researches of Dr. Stokes, Dr. Watson also published in the Medical Gazette, April 11, 1835, two cases of endo-pericarditis, in which he describes the *to-and-fro* sound of attrition, and perfectly distinguishes it from the co-existent valvular sound. M. Bouillaud does not appear to claim originality respecting the attrition sounds, but states that he had observed *bruit de souflet* in pericarditis at a period when he was completely ignorant of the labours of Drs. Latham, Hope, and Stokes."

tinguished from that of empyema or consolidation of the lung. It is by the preceding and accompanying circumstances that its nature is to be settled. By some it is objected, that the complication with disease of the lung or pleura acts in lessening the value of percussion in pericarditis. But this supposition is contradicted by experience. The combination of pericardial effusions with such affections is not common, at least in this country; nor, on the other hand, are those causes of pericarditis of frequent occurrence in which the effusion is so great as to simulate empyema. It is by connecting the results of percussion with the preceding and accompanying stethoscopic signs, that their real value can be established. If, for example, a dulness occurs within a short space of time, unattended with signs of pneumonia or of pleurisy, but having been preceded by friction signs referrible to the pericardium, no difficulty can arise in determining its nature. Again, pleuritic dulness almost always appears first posteriorly, while pericarditic dulness originates in the front of the chest. Now, although an empyema may cause dulness of the front of the chest, and a pericardial effusion dulness of the posterior portion, yet the following considerations will enable us to avoid error.

An empyema often causes dulness of the anterior portions of the chest. But this is, I believe, in all cases preceded by a loss of sound posteriorly. The rule, then, is this, that in cases where a doubt exists between a pleuritic and a pericarditic effusion, if we find the postero-inferior portion of the side clear, we are to adopt the latter supposition.

In both cases dulness anteriorly exists. In empyema the posterior dulness is antecedent, while in those rare cases of very copious effusion into the pericardium, sufficient to cause dulness laterally and posteriorly, the anterior dulness is the first to occur.

If, then, we find an extending dulness anteriorly, stretching from below upwards, not attributable to disease of the lung, and coinciding with a clear sound in the infrascapular region, we may make the diagnosis of pericardial effusion.

This dulness, in some cases, especially those where pericarditis is associated with diffuse inflammation, or some of the essential diseases, may be produced with great rapidity, and it may also disappear or diminish within short spaces of time.

I have not met with any of the cases of effusion so copious as to simulate empyema. In the case communicated by Dr. Corrigan, the distention of the pericardium reached to the first rib, and yet no difficulty seems to have been felt in the diagnosis.[1]

VISIBLE SIGNS OF ECCENTRIC PRESSURE.

The two most important observations on this subject with which I am acquainted, are those by Avenbrugger and Louis: the first relating to the production of an epigastric tumour; and the second to a dilatation of the side, analogous to that from empyema.

Avenbrugger's words are as follows: "Scrobiculum cordis tumor occupat, quem renitentiâ suâ distingues facilè a ventriculo flatibus turgente." This observation is confirmed by Corvisart, who cites a case in which seven or eight pints of liquid existed in the pericardium, causing not only obliteration of the natural hollow of the epigastrium, but producing a large tumour in that situation. This tumour appeared hard and resisting, and was occasioned by the yielding of the diaphragm before the pressure of the confined fluid. I have described a precisely analogous condition of the right ala of the diaphragm from an extensive empyema.

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[1] "It may be objected," says Louis, "to the value of percussion, that pericarditis is frequently complicated with pneumonia or pleuro-pneumonia, in which case it can be of no utility, since it would be impossible to say whether dulness proceeded from an effusion in the pericardium, or some other cause. The objection is a good one in cases of double pleurisy or pleuro-pneumonia, or where the disease occurs on the left side, but when these affections occur only on the right, percussion of the præcordial region has the same value as in simple pericarditis. Now, these cases are not very rare; out of seventeen cases of pericarditis, complicated with pneumonia, recorded by Morgagni, Corvisart, and Bertin, six are pleuro-pneumonia of the left side, five of double pleuro-pneumonia, and the remaining six of pneumonia at the right side, so that in a third of the complicated cases percussion would have been of the greatest utility. But in twelve of the thirty-six observations with which we are now dealing, there existed no complication with pneumonia or pleurisy, so that if we add these twelve observations to the preceding six, we have eighteen cases out of thirty-six, in which percussion would give the most useful results. It is not to be forgotten," he adds, "that I do not seek to place the results of percussion before the other signs of pericarditis, but only to estimate the value of the method, without which, no matter what may be the number and degree of the other symptoms, the diagnosis of pericarditis cannot be considered as certain."—*Recherches Anatomico-Pathologiques*, p. 280. See also Dr. Law's Pathological Observations, Dublin Journal of Medical Science, First Series, vol. vii. (1835.)

The next of these signs is that observed by Louis, namely, the dilatation of the præcordial region, which only differs from that in empyema by its remarkable circumscription. It was observed but in a single case, and the tumour extended from the hollow of the axilla to the edge of the false ribs; anteriorly and superiorly it ascended to within three inches of the clavicle. Over this tumour there was no œdema of the integuments, but pressure caused pain; and, as might be expected, there was perfect dulness on percussion, and absence of respiration over its whole extent. The sound of percussion over the remainder of the chest was natural, but the epigastrium and a portion of the left hypochondrium were dull. These parts were painful to pressure, and slightly prominent.

This observation was made on the eighth day of disease; the case was a very protracted one, nearly three months having elapsed before the patient's death; the pericardium contained a pint and a half (French) of fluid, which had depressed the diaphragm; the heart was somewhat diminished in size, and there was no evidence of any malformation of the chest.

Although no opportunity has occurred to me of observing this dilatation, yet I feel sure that it is not uncommon, and to this opinion Louis himself inclines. Perhaps, as Dr. Walshe has remarked, from the pericardium being less in connexion with the thoracic muscles than the pleura, dilatation of the side is not so constantly or so soon produced by its inflammation.

Finally, Dr. Graves has recorded an example of extrusion of the left lung upwards, in a case of pericarditis with extensive effusion. The patient, a child aged 10, was attacked with symptoms of pericarditis eight days before admission, and presented the usual signs of a pericardial effusion, with extensive dulness, indistinctness of the heart's sounds, and absence of murmur. The dulness extended from an inch below the left clavicle to the lowest part of the cardiac region, and to the middle and inferior parts of the sternum. The left side of the chest appeared fuller, particularly about the nipple, but measurement detected no inequality. On the following day a swelling of the lower part of the left side of the neck was evident, and on coughing a tumour was brought into view. The sound on percussion in the scapular region had a tympanitic character. The pericardium was found distended by serum to at least three times its natural size, and covered with lymph. After remarking on the extrusion of the left lung above the clavicle, Dr. Graves states his belief, that notwithstanding the equality of the sides on measurement, the pericardial region was really distended; and to this he attributes the increased resonance of the upper portion of the chest, on the principles indicated by Dr. Williams, of increase of tension causing augmented resonance.[1]

I have myself observed the displacement of the left lung to a considerable height above the clavicle, in a case of pericardial complicated with pleuritic effusion on the left side. In this case the tumour, though increased by coughing, was present for several days, and gave the pulmonary sound on percussion, with vesicular murmur and wheezing râle. The patient recovered. The tumour was so large as to produce during its continuance great deformity in the neck.

Having now examined into the physical signs of pericarditis, we proceed to consider its vital symptoms and history. Like many other local diseases, it is found in various forms and degrees of intensity, as shown by the amount of functional lesion, and the sufferings of the patient. Practically we may divide cases of the disease into three classes:—

1. Latent and trivial.
2. Latent and dangerous.
3. Manifest and dangerous.

The essential characters of the first of these classes are, localization, absence of essential disease, and lastly, a slight or feeble inflammatory action. We owe to pathological anatomy the discovery that almost every organ is liable to disease of this kind; disease, difficult or impossible to be recognised during life, because unattended by functional change, or any general disturbance. Occasionally, as in some cases of serous inflammations, it is accidentally discovered by physical signs. In pericarditis, if we admit that the milk spots are of an inflammatory origin, we must allow that the disease has affected a vast number of persons, yet in so mild a form, as not to excite suspicion at the time of its existence, nor to cause lesion in the function or structures of the heart. But when the number of instances are recollected in which not only a circumscribed spot, but even the whole pericardium has been attacked by inflammation, as shown by the stethoscope, yet without a symptom that would lead to a suspicion of the disease, we cannot hesitate to admit, that pericarditis is one of the most frequent of the unrecognised and often harmless diseases which affect the human body.

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[1] Clinical Medicine, vol. ii.

But we would commit an error if we supposed that the want of symptoms, and the feebleness of the physical signs, would justify us in considering the patient in a safe position. On the contrary, while any signs continue he must be carefully watched; for, in certain cases, a sudden change occurs, and the disease is converted from an apparently trivial and latent affection into a more severe form.

If we now consider the second class of these cases, namely, those which, though latent, are not without danger, we find that they may be divided into the complicated and uncomplicated forms. Of these, the first is, of course, the most important. We may have complication, as in cases where other serous inflammations are co-existing, such as pleurisy or peritonitis, when they occur as original local diseases; and, again, as in cases where the complication is with a general or essential disease, such as rheumatic fever. To this form it might be better to give the name of secondary latent pericarditis.

In latent pericarditis the disease is only discernible by physical examination, and as there is seldom any change beyond the effusion of lymph, the indications are limited to the tactile and acoustic friction signs.

It is this variety which is so often met with in rheumatism; and although in this disease the more severe forms may arise with or without endocarditis, yet the occurrence of the latent form is sufficiently common to justify the practical rule, that in any case of acute articular rheumatism we cannot be certain that the heart is safe unless by the careful employment of the stethoscope. So true is this, that it becomes absolutely necessary, if we seek to avoid being surprised by an attack of pericarditis, that we should examine our rheumatic patients from day to day, even though they present no symptoms of cardiac disease.

This liability to pericarditis, however, is less allied to the mere occurrence of rheumatic inflammation, considered as a disease of tissue, than to the essential state which we call rheumatic fever. It will be found that the liability to all the forms of carditis in rheumatism is in proportion to the severity and obstinacy of this fever. Indeed, in the apyrexial cases, even of acute arthritis, the pericardium commonly escapes; and in that remarkable disease of chronic-rheumatic arthritis, on which so much light has been thrown by the researches of Dr. Adams and Professor Smith, it rarely happens that the heart suffers, at least from acute disease. I have repeatedly observed this disease to affect a large number of the joints in a short space of time, and yet have found the circulation unaffected, and the heart, up to the last periods of life, free from any morbid acoustic sign.

As bearing on this point, and especially as illustrative of the necessity of considering rheumatic fever as an essential disease, not necessarily co-existing with arthritis, I may refer to two cases, one of which occurred to me in 1833; the other is given by Dr. Graves in his Clinical Medicine. In both, pericarditis preceded the inflammation of the joints, in my case by an interval of ten days, and in that by Dr. Graves, by five days. In the former case the symptoms were præcordial pain and oppression, with severe dyspnœa, and a cough which greatly aggravated the pain. The patient had also symptoms and signs of pneumonia of the right lung. It was not until the eleventh day that arthritis appeared, when the articulations of the lower extremities became swollen and painful, and this condition soon extended to the left arm. This patient sank with symptoms of pneumonia and pericarditis. In the case by Dr. Graves the symptoms and physical signs of pericarditis preceded the articular inflammation, and it was not until all signs and symptoms of pericarditis had subsided, that the patient was attacked with acute arthritis in the knees, shoulders, wrists, and ankles.

The disease ran the usual course of severe articular rheumatism, and lasted for ten or twelve days, during which time the heart, which was daily examined, exhibited no sign of disease. The treatment consisted in the exhibition of opium in large doses, as recommended by Dr. Corrigan, and succeeded admirably, none of the deleterious effects of the drug having been produced.[1]

To discuss the general pathology of rheumatism would be foreign to the objects of this work. With reference, however, to its connexion with pericarditis, we may adopt the following conclusions:—

1. That though the combination of pericarditis with acute articular rheumatism is common, yet that the disease of the heart is more closely related to the rheumatic fever than to the inflammation of the joints.

2. That the liability to pericarditis is in direct proportion to the violence and duration of the fever.

3. That in the apyrexial cases of acute arthritis, the liability to cardiac inflammation is but slight.

4. That pericarditis may be developed at any period of the disease, and even precede the arthritis.

5. That every variety and degree of pericarditis may occur in connexion with acute rheumatism, from the simple, dry, latent pericarditis, to the worst forms, combined with inflammation of the endocardium and muscular structure.

Although, as we might expect, the complication of acute rheumatism with pericarditis occurs under a variety of forms, yet three principal divisions of such cases may be made by the clinical observer. In the first, the disease, *as regards symptoms*, is truly latent, so that its discovery, which is only attainable by physical examination, is often accidental. In the second form, this latent disease may become manifest, and be indicated by a new train of symptoms, which at once draw attention to the internal disease, and it will then be found that the pericarditis has changed from the simple plastic form to a more severe affection, accompanied with copious effusion. This sudden change of dry, latent pericarditis into the more important forms of disease is an accident which must always excite great alarm.

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[1] On this subject Dr. Latham has the following important observations:—"But who shall say that endocarditis and pericarditis are not equally *essential* to it with inflammation of the joints, and that both are not equally derived from the attendant fever? . . . And I have seen a few cases (but very few) in which the inflammation of the heart has seemed to precede the inflammation of the joints. There has been fever, and with it palpitation and præcordial pain. Thus far the disease has been a puzzle. In a day or two the joints have become inflamed, and shown the disease to be rheumatism; and the endocardial murmur has been added to the palpitation and to the præcordial pain, and shown the sure existence of endocarditis from the beginning."—*Latham*, pp. 229, 232.

In the last form the invasion of the pericarditis is attended by distinct symptoms of cardiac suffering, and these, as Dr. Mayne has shown, may exist for one or two days before the appearance of any tactile or acoustic sign of the disease. Of the local symptoms, pain and weight in the region of the heart, with an increased impulse of the organ, are not uncommon. The pulse may, in some cases, be wiry and regular, while in others, irregularity of the heart's action is one of the first symptoms. It is important to notice this, as we may commonly connect the idea of irregularity of the pulse with the weakened state of the organ in the advanced stages of the disease. Evidences of irritation of contiguous organs are often seen. The left pleura may present symptoms of disease, bronchitic or pneumonic râles may appear in the left lung, while vomiting and epigastric tenderness indicate that the stomach sympathizes with the diseased organ, or itself partakes in the irritation. In some cases the invasion of these symptoms is attended with a mitigation of the arthritis, but this is by no means usual. I have been more than once led to suspect pericarditis from a sudden increase of fever, without corresponding increase of tumefaction in the joints. The countenance is anxious, with a sense of sinking about the heart, and apprehension of death.

In most cases the symptoms will be found attended by physical signs of attrition, of effusion, or both, varying according to the pathological state of the pericardium. The occurrence of bellows murmur is inconstant, and seems to indicate a complication with endocarditis.

We may now consider the general symptoms of the more severe forms of pericarditis, occurring independently of any rheumatic complication. On this part of the subject our best authority is Louis, who has accurately investigated the symptoms of this disease.[1]

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[1] Recherches Anatomico-Pathologiques, Art. Pericardite.

The system so long adopted by writers on medicine, of specifying a group of symptoms as indicative of a particular disease, has led to errors in diagnosis and practice. Hence, in attempting to describe or enumerate the symptoms of pericarditis, it must be understood that none of them are constant; and, further, that there may be great variation in the mode of succession of the phenomena in different cases. The first and most important symptom is pain in the region of the heart, frequently attended with a feeling of constriction or weight about the affected organ. This pain is generally less acute than that in pleurisy, but it is sometimes agonizing. It may also be felt in the epigastric and interscapular regions. Closely connected with this symptom is that of tenderness on pressure, with or without œdema of the integuments, in the cardiac and epigastric regions. In some cases the pain is intense and lacerating, and referred at first to the middle sternal region, attended with a most painful sensation of constriction of the chest.

Similar to the pain in pleurisy in its intensity, and in some cases in its seat, the pain of pericarditis has been occasionally observed to differ from that of pleurisy in this, that it is not augmented by a deep inspiration nor by change of position. It has been observed also to resemble that of angina pectoris in a remarkable degree. Thus, in a case by Andral, the patient was subject to dreadful exacerbations of pain, extending through the entire of the left side, accompanied by numbness of the left arm alternating with extreme pain. On three occasions the respiration became difficult, the pulsations of the heart tumultuous, the pulse imperceptible, and the surface of an icy coldness. On the subsidence of the paroxysm the heart's action would again become regular. In this case dissection discovered abundant concretions of coagulable lymph in the pericardium, and the sac itself was distended by a large quantity of bloody fluid.[1]

Pain, however, is frequently absent, or the patient complains only of uneasy sensations about the heart; and this may occur even when sudden and violent symptoms of another kind attend the invasion of the disease. Generally it may be stated, that the absence of pain is more likely to be met with in the complicated than in the simple cases, and the complication may either be with an essential disease, or some local affection.

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[1] Clinique Médicale, vol. i., Obs. iii., p. 15.

We are not yet fully informed as to the nature of the epigastric tenderness in this disease. Dr. Mayne observes that this symptom is generally very characteristic, and that it may be looked on as the most unequivocal general symptom of the affection. Out of eleven cases observed by him, it occurred in ten, and in five formed the principal source of the patient's suffering. It did not appear peculiar to any one period of the complaint. He observed that it was best marked when pressure was directed upwards and towards the pericardium, and that it was more circumscribed than the tenderness resulting from abdominal disease. Without, however, undervaluing these observations, we must not forget that in acute pleurisy of the left side, the epigastrium is often tender; and also, that from the rarity of acute gastritis we have seldom an opportunity of comparing the symptoms of that disease with those of pericarditis, which affection, so far as tenderness is concerned, may simulate inflammation of the stomach.

The next most important symptom is the difficulty of breathing, which is often attended by high and accelerated respiration. This latter character, however, may exist without the patient complaining of any dyspnœa. Louis attaches but little value to the oppression of respiration as a sign of pericarditis, although he admits that dyspnœa to a greater or less degree existed in all the cases which he has analyzed. He observes, however, that the symptom is of importance if it has supervened suddenly, and that no evidence of acute disease of the lung can be found. The same observation has been subsequently made by Dr. Mayne. Dr. Hope dwells on the dyspnœa in connexion with a constrained position, deviation from which produces a feeling of suffocation.

If with reference to dyspnœa we compare the diseases of pleuritis and pericarditis, the following difference may be noted. namely, that in pericarditis the tolerance of copious effusion is less often observed than in acute empyema. Indeed, in the latter affection it frequently happens, that after a certain period, not only the dyspnœa, but the acceleration of breathing disappears, so that the respiration is perfectly tranquil, at least while the patient is at rest. Such a condition, however, is rarely, if ever observed in copious pericardial effusions, and this can be easily understood if we consider the anatomical and physiological relations of the two diseases. In pleurisy but one-half of a double organ, as it were, is engaged, the opposite half remaining free to act; and the compressed lung may for a time be dispensed with. But in pericarditis we have not only the engagement of the entire organ by inflammation, but also its general compression, and in most cases a weakening or semi-paralysis of the muscle. Hence it is that although the subsidence of dyspnœa is often observed in empyema, even with copious effusion, it is so rarely met with in the analogous case of pericarditis.

Great stress has been laid on the character of the pulse in this disease, yet clinical experience establishes that no special condition of pulse can be described as belonging to any one form or stage of the affection. The following conditions may be met with:

1. Pulse small, rapid, and irregular at the onset of the disease, before the development of the ordinary physical signs.
2. The pulse becoming singularly slow at the very commencement of the disease.[1]
3. The pulse unaffected, except by the usual influence of fever. Under these circumstances it may be perfectly equal and regular. This is commonly seen in rheumatic fever with dry pericarditis.
4. Pulse regular, rapid, with a remarkable hardness.
5. Pulse regular, rapid, and feeble, while the action of the heart is excited.
6. Irregularity, inequality, and feebleness of the pulse, with a weakened action of the heart.
7. The same condition of pulse, with violent action of the heart.
8. The pulse may present alternations of regularity and irregularity.
9. Apparent suspension or obliteration of the pulse, succeeded by its re-appearance after a certain period.

On the symptoms of irregularity and intermission of the pulse, distention of the jugular veins, violet hue of the face, and coldness and œdema of the lower extremities, as occurring in the last stages, we need not dwell at any length. With respect to the first of these symptoms, however, it is to be remarked, that although Dr. Graves has observed it before any direct sign of pericarditis had occurred, yet in general we may hold it to be indicative of an advanced stage of the affection, when the heart is weakened, or suffering from inflammation of its muscles or lining membrane.

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[1] We owe this and the preceding observation to Dr. Graves. See his "Observations on Pericarditis," Clinical Medicine, 1848, p. 916.

There is a symptom in this disease referrible to the arterial system, which, though of great value, has been unnoticed, namely, an increased action of the cervical vessels. As to its actual frequency I cannot speak positively, but I have observed it in two remarkable cases, one of which will be given when we speak of the treatment of pericarditis. The patient was an adult. The second case was one of well-marked endo-pericarditis, occurring in a boy, under ten years of age. When this patient was first admitted he was in a state of collapse: the surface was pale, and the radial pulse extremely feeble, yet so violent was the action of the arteries of the neck, that it was visible at a distance, and drew immediate attention to the case. The physical signs were at first a double bellows murmur at the base of the heart, but on the fifth day a creaking friction sound, feeble with the first, but distinct with the second sound, could be heard at the apex. In a short time the friction signs became general, assuming their ordinary character, and still attended with valvular murmur. Liquid effusion now took place, while the increased action of the cervical vessels continued. At this period we observed that the friction sounds were most distinct when the patient sat up. This boy finally recovered, the disease having continued about eighteen days. Towards the close of the case the friction signs assumed a musical character, and were most distinct with the first sound.

In both these cases there was valvular murmur, and it is worthy of inquiry whether this increased action of the carotids may prove available in determining the presence or absence of endocarditis in such cases. But it may be laid down, that if the symptom be recent, and the constitutional state indicative of irritation or inflammation, this visible pulsation in the arteries of the neck, while the remaining vessels act feebly, should lead us to suspect some form of carditis.

Separately considered, this symptom, so far as I know, is met with in but four cases. It is noticed by Sir Astley Cooper as occurring in concussion of the brain, becoming evident when the patient sits up, and being then attended with increase in the frequency of the pulse.[1] It is met with in the earlier stages of permanent patency of the aortic valves, at which period it may be confined to the cervical vessels. We observe it, in the third place, in a curious and special form of chronic disease, which shall be presently described in full, attended with palpitation of the heart, increased action of the cervical arteries, and enlargement of the thyroid gland and the eye-balls. The fourth case is that just now specified, and hence, especially when the question of time is considered, the differential diagnosis will present no difficulty.

The risus sardonicus, contraction of the features, faintness, paleness, failure of animal heat, continued jactitation, insupportable distress and alarm, cold perspiration, and finally, from obstruction of the circulation, intumescence and lividity of the face and extremities, sometimes arising within the last twelve hours of life, are noticed by Dr. Hope as the most important symptoms of the disease in an extreme degree. To these he adds delirium and convulsions in the last stage.

Among the rarer symptoms in pericarditis authors have noticed the occurrence of maniacal excitement, sudden dissolution of the eye, and lastly dysphagia. The connexion of the first two of these conditions with pericarditis is doubtful, but the occurrence of dysphagia is, perhaps, more easily understood. Testa[2] has given some cases bearing on this point, which are worthy of careful study.

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[1] See Sir Astley Cooper's Lectures on Surgery. The symptom in question is given as diagnostic between compression and concussion of the brain. An increased pulsation of the carotids, analogous to that of the radial artery in whitlow, and, as I have observed in another place (see Researches on the Diagnosis of Aneurism. Dublin Journal of Medical Science, First Series, vol. v.) to those of the abdominal aorta in gastro-enteric fever, may be met with in cerebritis, but such a case could not be confounded with carditis.

[2] Testa's work, Delle Malattie del Cuore, was published in Bologna, in 1811, and dedicated to the Viceroy of Italy, Prince Eugene Beauharnais. The style of the author is extremely diffuse, but it is a work of great research, and contains many original observations, which, independently of their value as cases of cardiac diseases, are of importance to the student of this country who seeks to acquire an extended view of these affections as they occur in a warm climate and among another race of men. I published some extracts from this work, in an

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In the first case a man was attacked with high fever, dysphagia, and great difficulty of opening the mouth. Treatment had no effect on the symptoms till the sixth day, when the pulse moderated, but the dysphagia and pain in the throat remained. Two days after this, appeared a swelling of the right parotid region, which rapidly subsided. He died on the tenth day. The fauces presented not the slightest trace of inflammation; the pericardium was thickened and hardened, and the sac filled with fœtid sanies in great quantity. The heart showed marks of severe inflammation, both of its membranes and muscular structure, and the ventricles were lined with lymph. The diaphragm, liver, and upper portions of the stomach were inflamed, as were all the vessels, venous as well as arterial, in the vicinity of the heart. There was a slight degree of pleurisy.

In a second case, a woman long subject to a quartan fever, followed, after its cure by bark, with violent tremors of the lower extremities, was attacked by rigors, succeeded by intense heat and severe pain in the fauces, and the greatest difficulty in swallowing. On the fourth day she was conveyed to hospital; her face was deep red, her parotids swollen, and the tonsils of a bright red colour. Respiration was difficult, and similar to that of persons affected with angina; her voice was low and feeble; there was no cough, but no substance, solid or liquid, could be swallowed. The diagnosis was made of angina pharyngea, with some laryngitis. During the last two days she had alternations of coma and delirium, and during the latter she swallowed with less difficulty; the pulse was small and tremulous. She died on the seventh day.

This case was a combination of severe pericarditis with pleurisy. The pericardium was thickened, and contained a great quantity of whitish purulent fluid; the heart had likewise suf-

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English dress, in 1889, with reference to those cases of carditis which simulate affections of the throat (see Dublin Journal of Medical Science, First Series, vol. xiv.) In reference to these cases Testa observes: "Nessuno per altro, ch'io sappia, à fatto finora distinta menzione dei sintomi anginosi, li quali non solo si uniscono ai segni proprii del cuore inflammato, ma bensi li nascondono quasi affatto sotto il solo apparecchio anginoso."—Vol. iii. p. 106. The chapter containing these cases is headed 'Dei Pericarditici e Carditici Anginosi."

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fered from carditis. Two more cases of dysphagia in connexion with disease of the heart and pericardium are given by the same author, the symptoms in one being a severe smarting in the œsophagus whenever the patient was tempted to swallow even a mouthful of water. Neither in the internal nor external fauces could any alteration be found. These symptoms attended by fever and difficulty of breathing, occasional delirium, and an intolerable sensation of burning heat in the thorax, extending from below the xiphoid cartilage to the fauces, continued up to the period of death, which took place about the seventh day. The pulse was small, rapid, occasionally intermitting, and the patient, an adult male, was constantly exposing his chest, being unable to bear even the lightest covering. He was extremely restless, and troubled with spectral illusions. False membranes were found on the pleura, and the sac of the pericardium contained a great quantity of thin sanies; the heart, somewhat hypertrophied, was ulcerated on its surface. Marks of inflammation were found on the diaphragm and in the liver, which was enlarged.

The last case is an example of pericarditis with serous effusion, in which the symptoms were fever, a deep burning pain in the chest and fauces, with dyspnœa, pain in the left arm, and a soft, irregular pulse.

I have observed dysphagia as a symptom in thoracic inflammations, and its accompanying phenomena seemed to prove that it was less the result of any mechanical condition, such as pressure on the œsophagus, than of some excited irritability either of that tube or of parts immediately in contact with it.

A woman, aged upwards of 60, of an extremely spare habit, was attacked with symptoms of acute lumbago after exposure to a draught of cold air. She remained for three or four days without paying attention to these symptoms, when the pain suddenly left the loins and ascended to the interscapular region. When I saw her, the breathing was hurried; the pulse small and wiry; and she complained of an extraordinary sensation upon attempting to swallow. As the mouthful of food or drink passed down a few inches below the pharynx, it excited a feeling of tearing or burning through the remainder of the passage, which immediately subsided on the ingesta reaching the stomach. There was no regurgitation, but her sufferings from the dysphagia were extreme.

On examination I found the lower portion of the left side sounding dull on percussion, with well marked ægophony at the root of the lung, extending laterally for two or three inches. The action of the heart was rapid, but not irregular, nor were any of the direct signs of pericarditis present.

On the next day the heart was evidently displaced, and pulsated strongly under and to the right of the sternum, while it was scarcely perceptible in its natural situation. In the course of this case the action of the heart became very irregular, but no other symptom of disease of the organ was manifested. After several relapses of the pleuritis, the effusion was absorbed; but on each exacerbation the dysphagia became greatly aggravated, and was always relieved by the application of leeches over the affected portion of the left side. After her recovery from the pleuritis the irregularity of the heart continued.

In two cases of pneumonia I have observed symptoms somewhat allied to those described by Testa; I have known aphonia, without any other sign of laryngeal disease, to set in and subside with an extensive inflammation of the left lung. The case was that of a gentleman of full habit, who was attended by Dr. Graves and myself. The hepatization resolved with extreme slowness, but as soon as the side had recovered its sonoriety, the aphonia disappeared. This was a most insidious case.

In a young man attacked with pericarditis the voice underwent a great variety of changes of tone, and was not restored for several weeks, when all symptoms and signs of pericarditis had subsided. In this case, the liquid effusion was never very considerable. The phenomena were slight dulness, with various modifications of the rubbing sounds.

The foregoing facts all seem to prove that the symptom in question, however produced, is less a mechanical than a vital effect. It occurs in the earliest, sometimes in the very first periods of the case, and at a time when but little distention of the pericardium has occurred. It may disappear in the more advanced periods, and may be accompanied with pneumonia, indicating interference with the functions of organs placed out of the reach of pressure. Finally, when we consider its rarity in hydro-pericardium, and in cases of empyema with great eccentric displacement, we must, I think, adopt the above mentioned view of this curious symptom.

I have already alluded to a case of sudden pleuritic and pericardial effusion, in which the singular phenomenon occurred of *the thrusting upwards of the lung, so as to form a very large tumour above the clavicle.* This tumour had a puffy, elastic feel, and the stethoscope detected evident vesicular murmur over its surface. The disease was subdued by active treatment, and in a few days the tumour disappeared. Here the left lung was suddenly compressed by the double effusion, and yet no dysphagia was observed.[1]

But with reference to the cases from Testa, this question arises: were they examples of primary disease, or in reality instances of diffuse inflammation with or without phlebitis, and inducing the pyogenic state? There are strong grounds for believing that such was their real character, and that the pericardial disease was but one of a group of secondary lesions. Let us remember the occurrence of parotid swellings, of the fœtid, sanious, and purulent effusion into the pericardium, the evidences of inflammation in the diaphragm, pleura, stomach, and liver, to say nothing of the great vessels in the vicinity of the heart; and lastly, the symptoms of nervous disturbance, and we cannot but suppose that these cases were of a phlebitic nature.

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[1] In the works which I possess on diseases of the heart (with the exception of Testa,) I have not been able to find any notice of dysphagia as a symptom of inflammation of the pericardium or pleura. I have examined carefully the works of Senac, Corvisart, Bertin, Laennec, Bouillaud, Hope, and Andral. Testa alludes to the case of the wife of Polemarchus, recorded in the fifth book of the Epidemics, but I consider it as scarcely one in point. That of the courier in Morgagni is more important, and I shall not apologize for introducing it.

"Vir erat annorum amplius quadraginta, qui Foro Cornelii Bononiam identidem ventitabat pedes, res traditas huc illinc, et vicissim hinc illuc ferens. Is cum sæpe vel ab itinere calens, biberet, postremo præsertim tempore quo assidue sitiebat, rheumate ad fauces gravi, et febre correptus, in Nosocomium admissus est. Mox ibi de faucibus non amplius conquestus, suum in ventre morbum omnem esse dicebat; nulla tamen de re querebatur magis, quam de Spinæ ad lumbos dolore, quo ea sibi media dissecari videbatur. Erant propterea qui intestinorum inflammatione laborare hominem, crederent: VALSALVA autem in thorace eam esse, suspicabatur. Erat autem pulsus debilis, humilisque; sed qui tamen ligatus, ut ajunt, videretur. Surgere, quasi abiturus, sæpe voluit. Per hæc intra tertium, an quartum ex quo in Nosocomium venerat, diem confectus est. VENTER nihil habuit quod secundum naturam non esset. In Thorace autem ab altera potissimum parte humor stagnabat, in quo frusta natabant, quasi membranularem albissimarum; ut nihil magis referret, quam serum vaccinum, particulas retinens casei secundarii. Pleuræ vasa magis quam solent, rubebant, nec multo id tamen. *Pericardium vero fuit adeo distentum, ut vix compunctum, aquæ ejus qua erat plenissimum, tenue quasi filum ad non modicam altitudinem ejacularerit.* Cordis mucro plus æquo rubens, leviter inflammatus fuisse videbatur."—Lib. ii. *De Morbis Thoracis, Epist. Anat. Med.* xvi. Art. 40.

Taking this view of the matter, it may be asked whether the dysphagia might not have proceeded from an inflamed condition of the great network of veins which ramify in the retro-pharyngeal cellular structure. Professor Smith has observed, that in many cases of diffuse inflammation these vessels are so much affected as to cause suppuration throughout the whole of this loose reticular tissue, from the pharynx down into the mediastinum, and that this condition may exist without our ever being able to discover any tumour in the pharynx, although dysphagia and other symptoms of angina are present.[1]

Among the rarer symptoms of, or rather the accidents connected with this disease, are we to place the sudden destruction of the eye, as described by Corvisart. This author gives a case of pericarditis in which not less than two pints of sero-purulent fluid were effused, while the heart was covered with a thick albuminous layer. The disease was singularly uncomplicated, and had apparently resulted from a blow on the cardiac region. The general symptoms presented nothing remarkable except the spontaneous and almost sudden dissolution of the right eye, without any preceding or accompanying inflammation. At the time of this occurrence the patient was in a state of great prostration. In another case of pericarditis, which terminated in adhesion, the right eye became ecchymosed and inflamed during an access of cardiac suffering, but no dissolution of the organ is reported to have taken place. In his first case the most careful examination failed to detect any cerebral disease, and the sudden dissolution of the eye remains an unexplained fact.

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[1] See an important paper on the subject of abscesses posterior to the pharynx by the late Mr. Carmichael. (Transactions of the Association of the College of Physicians of Ireland, vol. iii.) Also, the Elements of the Practice of Medicine, by Drs. Bright and Addison; and a Memoir on Pharyngeal Abscesses, by Mr. Fleming. (Dublin Journal of Medical Science, First Series, vol. xvii., 1840.)

Corvisart refers to Testa for examples of loss of vision in connexion with disease of the heart; but the cases in question have nothing in common with the instance given by the French pathologist. One of these cases appears to have been an example of amaurosis; others of superficial or deep-seated inflammation, and there is nothing to lead to the belief that the injury of vision was induced by disease of the heart.

It is more than doubtful that the sudden dissolution of the eye, as noticed by Corvisart, is to be considered as depending on carditis, or any form of disease of the heart; the eye suppurated, and gave way without previous inflammation.[1] Such an accident, resulting from disease of the heart, has never, so far as I have seen, been observed in this country; but that it occasionally occurs in cases of purulent phlebitis is certain. In this condition, and without the slightest previous distress, as referred to the eye, the patient sometimes becomes suddenly blind of one or of both eyes. Within a short time pus can be detected in the chambers of the organ, and should the patient survive sufficiently long, the coverings may give way, and collapse of the eye-ball follow from the simultaneous evacuation of the humours and purulent secretion. The history of Corvisart's case seems to bear out the view that some typhoid condition of the system existed, and it is remarkable that not less than sixteen days elapsed between the infliction of the blow and the appearance of fever and oppression of the chest.

We cannot, then, as I conceive, admit the sudden dissolution of the eye as one of the symptoms of pericarditis. In the present state of our knowledge it is only met with in phlebitic and other analogous forms of inflammation.

We shall presently have to examine a special form of disease of the heart, which is attended with a peculiar, but very different condition of the eye.

Having now considered the signs and symptoms of pericarditis, we may, with advantage, study some examples of the disease.

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[1] See the work of Corvisart, p. 17.

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CASE I.—*Acute dry Pericarditis, following the disappearance of a cutaneous disease; production of the Leather-creak Sound within a short time before death.*

A boy, aged five years, had been cured of a cutaneous disease, the nature of which was not ascertained. In a few days he became ill, with symptoms of inflammatory fever; he had thirst, occasional vomiting, short cough, hurried breathing, and orthopnœa; the left side of the abdomen was full and tender, and he complained of pain, referred to the belly. When I first saw him he was sitting in bed, his legs drawn up, and with hurried, high, and laborious respiration. The lips were livid, the face œdematous, and the jugular veins distended; pulse 130, small, jerking, but regular. The impulse of the heart was violent, with a distinct rubbing sensation communicated to the hand; a very loud friction sound attended both sounds of the heart, and was heard to the right of the sternum, under the clavicles, and along the spine. In the latter situations, however, it had lost much of its roughness, and approached to the bellows murmur. The sound on percussion over the heart was dull to an unusual extent, and the respiratory murmur everywhere puerile and pure, with the exception of a slight and fugacious bronchial râle. He died on the third day after admission into hospital. On the day before death the jugular veins pulsated, the abdominal tenderness had greatly increased, *and the friction sounds assumed the character of the "bruit de cuir neuf" of Collin.*

On dissection, a general hypertrophy of the heart was discovered; the pericardium was thickly covered on both surfaces with a reticulated layer of reddish coloured lymph; no adhesion had taken place, nor was there any liquid effusion into the sac; the mitral and aortic valves were slightly thickened, and somewhat opaque, but otherwise healthy. Circumstances did not permit examination of the remaining viscera.

The occurrence of a dry pericarditis attended with such violent symptoms as were observed in this case is worthy of note. It is rare to meet this form of the disease unless as a mild affection, and it is probable that to the previous existence of disease of the heart we are to attribute the great virulence of the attack. We shall just now examine another case, in which a dry pericarditis, co-existed with hypertrophy, and in which also the symptoms were unusually violent. It may be laid down, that where we have considerable dulness over the heart, with a friction sound extending over a large portion of the chest—a friction sound which does not diminish, as where liquid is effused, but which, as in the case now before us, actually increases in intensity with the advance of disease—we may determine that the case is one of dry pericarditis engaging an hypertrophied heart.[1]

CASE II.—*Acute dry Pericarditis with Hypertrophy and Dilatation of the Heart.*

A man, aged 20, after recovering from an attack which resembled gastric fever, but was attended with severe pain in the lower sternal region, was soon afterwards admitted into hospital with the following symptoms, which were of four days' standing:—Fever of an inflammatory type; pulse small, weak and rapid; hurried and difficult breathing; great tenderness of the surface, and pain in the lower portion of the chest. With the exception of dulness on the anterior portion of the right side, there was no physical sign of thoracic disease observable. Next day the pain was fixed in the lower portion of the right side; the respirations were 48 in the minute, and the pulse irregular. The breathing soon became completely thoracic, yet no sign of pulmonary disease could be detected. On the day before his death he was seized with a violent stitch in the left mammary region. The intermissions and irregularity of the pulse increased, and for the first time intense rubbing sounds were discovered over the heart, attended with distinct friction sensations communicated to the hand. His death took place on the eighth day of the attack.

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[1] Referring to Dr. Graves' observations on the extension of the sounds in pericarditis, it will be remembered that he dwells on the co-existence of an enlarged heart as an important cause of the occurrence. I have already expressed my conviction that the extension of the sounds has more to do with their nature than the amount of surface of the heart from whence they proceed. I did, however, in my observations on this case, published in 1834, suggest that the enlargement of the heart might be a cause of extension of sounds. My words were as follows: "The dulness of the region of the heart was satisfactorily accounted for by the great hypertrophy of the organ; a circumstance which, taken in connexion with the excitement of the heart and the age of the patient, may explain the unusual extent to which the stethoscopic phenomena of pericarditis were audible."—*Researches on the Diagnosis of Pericarditis, Dublin Journal of Medical Science*, First Series, vol. iv., 1834.

The best and most comprehensive account of pericarditis as occurring in infancy and childhood is to be found in the work of Dr. Churchill on the Diseases of Children. It does not appear that when the affection is met with in young children there is any special character attending the disease. Its symptoms, signs, and pathology, are the same as are met with in the adult. Several cases of latent pericarditis are recorded, but we cannot say that this latency is more common in the child than the adult. It is, however, probable, that in its uncomplicated forms the disease is more often latent in the child and infant. Dr. Lees has given an example occurring at the age of four months. The disease was exceedingly obscure. The infant looked ill, and seemed to suffer severe internal pain. Death occurred after long-continued convulsions, and the only morbid appearance found was a thick layer of greenish lymph, spread over both surfaces of the pericardium. In this case there was no cough, nor impeded respiration. (See the Transactions of the Pathological Society of Dublin for January, 1841.) The work of Billard, "Maladies des Enfans," may be consulted; also, the great work of Cruveilhier.

On dissection, the heart was found greatly enlarged and extending to the right side, so as to displace the lung. The pericardium presented evidences of chronic and of acute disease. A cartilaginous band, of nearly an inch in width, connected the heart a little above the apex with the outer fold of the pericardium, and the whole of the internal surface of the sac had a mammilated appearance, produced by depositions of a semi-cartilaginous consistence, super-imposed on which was a layer of soft lymph, of a deep red colour. The valves were healthy, and no change beyond cadaveric engorgement was found in the lungs.

The true nature of this case was not discovered until the day before its fatal termination. It was the first in which I ventured to make the diagnosis of pericarditis from physical signs, and it furnished the basis of subsequent investigations.[1] This case occurred in 1830. It has been already alluded to in the present work, as bearing on the question of the effect of enlargement of the heart in causing that extension of pericarditic sounds which may lead to their being mistaken for the signs of diseased valves. There is every probability that there were two attacks of pericarsembled gastric fever, but was attended with severe pain in the lower sternal region, was soon afterwards admitted into hospital with the following symptoms, which were of four days' standing:—Fever of an inflammatory type; pulse small, weak and rapid; hurried and difficult breathing; great tenderness of the surface, and pain in the lower portion of the chest. With the exception of dulness on the anterior portion of the right side, there was no physical sign of thoracic disease observable. Next day the pain was fixed in the lower portion of the right side; the respirations were 48 in the minute, and the pulse irregular. The breathing soon became completely thoracic, yet no sign of pulmonary disease could be detected. On the day before his death he was seized with a violent stitch in the left mammary region. The intermissions and irregularity of the pulse increased, and for the first time intense rubbing sounds were discovered over the heart, attended with distinct friction sensations communicated to the hand. His death took place on the eighth day of the attack.

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[1] Researches on the Diagnosis of Pericarditis, Dublin Journal of Medical Science, First Series, vol. iv. (1834,) Case I.

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[1] Researches on the Diagnosis of Pericarditis, Dublin Journal of Medical Science, First Series, vol. iv. (1834,) Case I.

This case has been already referred to in the present work, as showing that even with an hypertrophied heart the sounds of friction may not extend beyond the limits of the organ.

We would gain little by dwelling on cases of uncomplicated pericarditis, the characters of which are now so well known. Let us rather study the disease in its combination with other affections.

But before entering on this part of the subject, we must refer to some observations by Dr. Mayne, which show that an inflammatory effusion may take place into the pericardium, and yet no friction sound be developed. Cases of this kind are rare, and the want of friction signs depends on the nature of the secretion and the smoothness of the surface. It will be remembered, that in the instance recorded by Dr. M'Dowel no friction sound was discovered. The heart was bathed in purulent matter, so that from the moment of the formation of the fistula we may suppose that a fluid of great lubricity covered the organ. Further, it is probable that in some of the sub-acute cases, with an effusion almost purely serous, there would be no friction, unless, perhaps, when the surfaces came into contact on the absorption of the liquid.

Dr. Mayne has given two cases in which friction signs were not developed. In one, effusion into the pericardium was found on the first examination sufficiently extensive to cause dulness of the region of the heart. The patient, a woman, was then forty-eight hours ill, but it is probable that had she been seen at the onset of the disease some friction phenomena would have been discovered. The symptoms were, irregular action of the heart, with an exceedingly weak and sometimes imperceptible impulse. Both sounds could be distinguished, but without any friction or bellows murmur. The pericardium was found greatly distended with liquid of a sero-purulent character, and a similar effusion existed in both pleuræ. Fragments of false membrane existed upon the surface of the heart.

In the second case it is more than probable that friction signs were never developed. It was one of acute anasarca, succeeded by diffuse inflammation of the cellular membrane of the neck, chest, and abdomen. The parts affected were exquisitely tender, the pulse very rapid and small, and the fever well marked and of a typhoid type. The patient complained of slight uneasiness about the heart, but nothing peculiar was discovered by the stethoscope. The action of the organ was very rapid and weak, but there was no *frottement*, or other unnatural sound. Death took place on the second day of the diffuse inflammation; and on examination the pericardium was found to contain seven or eight ounces of thin pus. There were no false membranes. The pericardium presented some vascular patches.

Dr. Mayne observes, that the stethoscopic signs of pericarditis were never developed in this case, which he accounts for by the fact that no lymph had been secreted.[1]

We may now proceed to examine some instances of pericarditis occurring in combination with other affections, both local and general.

CASE III.—*Acute Pericarditis with Pneumonia and Arthritis.*

A man, aged 35, was admitted on the tenth day of his illness, with symptoms of severe pneumonia complicated with arthritis. He had been first attacked with pain and oppression at the præcordia, with severe dyspnœa and cough, followed in the course of twenty-four hours by articular inflammation in the lower extremities and left arm. On admission he appeared moribund; his countenance was sunken and anxious; he had laborious respiration, with frequent cough, attended with muco-purulent expectoration, which the night before had been tinged with blood; the knee-joints were inflamed and painful, and he had dull pain at the lower portion of the sternum, increased by coughing and by pressure at the epigastrium; pulse 96, feeble, small, but regular; the chest sounded well anteriorly. There was some dulness over the inferior portion of the right side, and here, as well as in the corresponding part of the left side, an intense crepitating râle was manifest.

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[1] See Dr. Mayne's observations on Pericarditis, Dublin Journal of Medical Science, vol. vii. Though we admit that in cases of sub-acute pericarditis with an almost purely serous effusion, and again in others where a purulent fluid is produced from the first, friction signs may be absent, yet these are exceptional cases, and their occurrence furnishes no argument against the utility of physical diagnosis in this disease. When we recollect that friction signs and dulness may coincide, and that unless in a case observed from its very commencement, we cannot absolutely say that friction signs never occurred, it will appear plain that the number of instances in which these phenomena were absent will be found to be exceedingly small.

The sounds of the heart were peculiar, and varied remarkably with the position of the stethoscope; when applied over the left side of the heart, the pulsations were found to be accompanied by a sound resembling an indistinct *bruit de râpe;* but along the lower part of the sternum there was an exceedingly loud and perfect friction sound, which accompanied both the systole and diastole of the heart. Towards evening the patient, after having taken some stimulants, was found in a state of general re-action. On the following day the pulse was 88 in the minute, perfectly regular, and somewhat contracted; he said he had no pain in the lower part of the sternum, except when he coughed; the impulse of the heart was natural, and the lower part of the sternum continued clear on percussion.

The *frottement* and simulated *bruit de râpe* continued as yesterday, but a new and remarkable phenomenon was observable: every four or five beats a change of character occurred with great regularity, constituting a most perfect rhythm. This was found to be connected with the respiratory movements, the sound being roughest and most intense during inspiration, but during expiration becoming feebler, and more like the bellows murmur. On the following day, the 12th, we found that the phenomena of the heart were distinctly modified, as compared with the day before; the rasping sound being now distinct at the left side of the heart, and wanting at the right, where a double bellows murmur was audible: the distinctness of which was, as before, modified by the action of respiration.

Three days having passed, we could still feel a slight fremitus over the heart, the rasping character of the friction sound had disappeared, and the region of the heart sounded clear. The next day the harsh rubbing sound had completely disappeared, the sound being a pure double bellows murmur. At this time the patient's general state was greatly improved; in a few days, however, the pulmonary symptoms re-appeared, with an increase of the phenomena of the pericarditis; some time after this he sunk. We were not able to obtain a dissection.

The treatment consisted of local bleeding, counter-irritation, and the use of colchicum and mercury.

In this case, although we cannot appeal to the results of dissection, yet I would submit, that there can be but little question as to the nature of the disease and the physical alterations of the pericardium. This was obviously a case of dry pericarditis: the patient, as happens in many instances, laboured under a complication of disease; the right lung being severely affected, and the articulations the seat of an obstinate inflammation. We may hereafter inquire how far this circumstance of complication may serve to explain the occurrence of that variety of pericarditis in which lymph alone is effused. It is at all events remarkable that in most of the cases of this disease that I have witnessed, the patients laboured under inflammation in various organs and in different tissues.

This patient presented stethoscopic phenomena perfectly analogous to those observed in the former cases, where we had an opportunity of verifying our diagnosis by dissection. The sound on percussion over the heart continued clear, and the impulse of the organ was always distinctly perceptible, and accompanied by a rubbing feel; circumstances tending to show the non-existence of liquid in the cavity of the pericardium. During the progress of the disease we observed those remarkable changes in the character of the sounds which I have noted in the preceding cases: the passage of the rough rasping sound, to one giving the idea of a smoother surface; the first similar to the *bruit de râpe*, the second to the bellows murmur. But in this case two other circumstances of importance are to be noted.

First, the change of situation of the rasping sound. It will be recollected that at first this was most distinct at the right side of the heart, but that shortly after it became evident at the left, where previously a sound similar to a double bellows murmur was only audible. This I look upon as a circumstance of great importance in the diagnosis between this disease and affections of the valves. It may happen, as I have often myself observed, that in cases of extensive valvular disease the rasping sound may pass into a bellows murmur, in consequence of the moderated action of the heart, the result of rest or treatment. On excitement taking place, however, the original sound will be restored. But here we have a change, first in character, and secondly in the actual situation of the sound, a circumstance easily explicable by the extension of the disease and the modifications produced in different portions of the pericardium. The slight extent to which these sounds are audible, unless during great excitement, gives additional weight to this explanation. I do not know of any case of valvular disease in which the rasping sound was observed, in the course of twenty-four hours, to change from the right to the left side of the heart.

Secondly, the modification produced in the sounds of friction by the action of respiration. It will be recollected that the rubbing sounds became more distinct, and conveyed the idea of a rougher surface during inspiration; during expiration they became less distinct, and closely approached to the bellows murmur. We found further, that if the patient held his breath, the character of the sound was between these two extremes, and that the peculiar rhythm ceased, evidently showing that it was produced by the action of respiration.

CASE IV.—*Acute Arthritis; Pericarditis; double Pleuro-pneumonia; recovery.*

Frances Kelly, aged 24, of a vigorous constitution, was attacked on the 25th of March, 1833, with symptoms of severe arthritis, affecting most of the articulations. She had considerable inflammatory fever, but no pain whatever in the chest. Previous to this illness she had enjoyed the best health. In the course of six days she was admitted into the Meath Hospital, where I found her labouring under a general arthritis, although none of the joints were in a state of excessive inflammation. She had high fever, and a full, strong, and perfectly regular pulse,* no pain of the chest, cough, or dyspnœa. The heart's action was strong, *and a slight friction sound was audible near to the apex.*

Free bleedings, both general and local, were ordered. The tartar emetic treatment was pursued for nearly five days, when we had to desist from the occurrence of vomiting and purging.

On the seventh day after her admission I found her in a state of high fever, and complaining of severe pains in the joints, which, however, did not show any corresponding increase of inflammation. The pulse full and hard, 130 in the minute; respiration 40. The increase of fever, without increase of arthritis, led me to suspect some severe visceral inflammation, and I directed my attention to the heart, but could not discover any unequivocal sign of disease.

Next day, however, there was decided evidence of the existence of inflammation both in the pericardium and left lung. The left side of the chest in its lateral and inferior portions sounded dull, and the respiratory murmur had become feeble generally. In addition to this, a decided pleuritic *frottement* could be heard in the antero-inferior portion. That it proceeded from pleuritis was obvious from this, that it was synchronous with respiration, and whenever the patient held her breath the sound altogether ceased.

The sounds of the heart were accompanied by a loud rasping, occurring with both sounds. This was very loud at the base of the heart, and scarcely audible at the apex. Under the clavicle, and in the posterior portions of the chest, the sound was inaudible, although the pulsations of the heart were distinctly heard. No fremitus was perceptible. Her countenance was extremely anxious; she declared that she had no pain in the chest, but had a sensation of sinking about the heart, with distressing palpitation; great prostration, but no syncope; she was apprehensive of speedy death; respiration hurried, but not difficult; pulse 124, hard and thrilling, but regular. She had slept badly, and begged for a narcotic. Leeches, calomel, and digitalis.

On the next day, although there was an evident improvement in the general symptoms, the rasping sound had extended over the whole region of the heart. The following is the report of the 10th:—

The anxiety and sense of sinking are much diminished: breathing easier; pulse 110, soft and full; impulse of the heart less;

urine scanty and high-coloured; no mercurial action. The friction sound continues distinct over the whole region of the heart, but has lost much of the roughness, and passes into bellows murmur; the left side still sounds dull. No examination was made of the posterior portions of the chest.

11th. The rasping sound was found to have ceased at the apex, but it still continues at the base of the heart with evident fremitus. Under both scapulæ a distinct pulmonary friction was audible, and the right side had become dull on percussion. Blister, mercurial frictions.

12th. General improvement; the friction sensation of the heart had nearly disappeared, being only perceptible at the sternal end of the third rib. No change in the pulmonary signs.

13th. All friction sensation had disappeared from the heart, but from our unwillingness to disturb the patient, no examination was made of the posterior portions of the chest. No ptyalism had been produced.

14th. The patient was not so well. The disease in the lungs showed but little disposition to resolve, and the rasping sound reappeared at a point which could be exactly covered with the stethoscope over the right side of the base of the heart. It was heard nowhere else: there was neither rasping nor the simulated bellows murmur on any other portion of the heart. I now determined to leech the right side freely, and to again try the tartar emetic treatment, particularly as throughout the case the appetite had continued good and the tongue generally clean. She used the remedy for six days, at the rate of six grains each day, with gradual improvement in the pulmonary symptoms. The region of the heart, however, became extensively dull, the rasping sound continuing at its base. The dulness gradually subsided, and on the 22nd of April the sound over the heart was perfectly natural, and the pulmonary congestion nearly removed. The following is the report of the 24th:—

The phenomena of the heart are now perfectly natural. There is still some dulness over the posterior and lateral portions of the right side, with some friction sound.

In a few days this patient was quite convalescent, and the most minute examination of the heart could detect no departure from the state of health.

Let us now consider this disease under certain pathological and mechanical conditions.

CASE V.—*Pericarditis supervening on acute Empyema of the right Side; Protrusion of the Diaphragm, and Displacement of the Liver.*

Patrick Murphy, aged 40, was admitted into the Meath Hospital on the 22nd of March, 1833. On the 15th (seven days before admission,) he was attacked by a rigor, followed by acute pain in the right side. On admission, he complained of a severe stitch in the right side, aggravated by coughing and inspiration; his expectoration was scanty, and consisted of mucus and serum; respirations 54 in a minute; pulse 106, small and hard; tongue very foul, with redness at the edges and tip; thirst, and epigastric tenderness.

On percussion we found that the right side, both anteriorly and posteriorly, sounded dull, particularly in its more inferior portions, where the integuments were exquisitely tender. This side was also found an inch larger, by measurement, than the other, and no vibration was communicated to the hand when the patient spoke, though this was distinctly felt in other parts of the chest. Respiration over the superior portions of the chest was heard feebly, and we observed a doubtful ægophony under the scapula. The liver was observed to extend about an inch below the ribs, forming a tumour exquisitely tender on pressure; decubitus on the affected side.

Active treatment was adopted, the patient was bled generally and locally, and calomel and opium were exhibited in free doses, but no effect appeared to be produced on the disease, as on the 24th the dulness was found to have extended, the side still more dilated, and the intercostal spaces elevated. On the 29th we found that both sides corresponded in measurement, yet there was no appreciable improvement in the other symptoms; no satisfactory mercurial action had been induced, although the patient had been daily using mercury. On the following day it was observed that the dulness extended quite across the sternum, and the respiration in the superior portion of the lung had assumed a bronchial character. We also observed, for the first time, a well-defined sulcus existing between the false ribs and the superior portion of the hepatic tumour. On the 31st it was found that the patient had suffered greatly from orthopnœa during the night, and at the hour of visit he could scarcely breathe in the recumbent posture. The hepatic sulcus was more defined, and the liver evidently pushed towards the left side; respirations 40; pulse 92, small, feeble, but perfectly regular.

On examination, I found that the region of the heart sounded clear on percussion; its impulse could be distinctly felt; and evident fremitus was communicated to the hand when placed over the cardiac region. The action of the heart, though rapid, was perfectly regular, and a morbid sound between that of the *craquement de cuir neuf* and *bruit de râpe* was distinctly audible. The patient declared he had no pain whatever in the region of the heart, but stated that during the last two days he had felt some slight uneasiness in that situation. On the next day he was obviously sinking, there was some delirium, and the pulse for the first time became intermittent. We observed that the hepatic sulcus, which for the last two days had been so well marked, was now nearly imperceptible; the sound of friction continued the same as on the day before. The patient died shortly after the hour of visit.

Dissection.—On opening the abdomen, the thin edge of the right lobe of the liver was found to descend as low as the umbilicus, the left lobe extended into the corresponding hypochondrium, and the horizontal fissure was nearly in the direction of the median line, though inclined slightly across it. The hepatic tissue was soft, and of a red colour, and we observed that the sulcus between the under surface of the diaphragm and the upper portion of the liver was very inconsiderable.

On removing the liver, its diaphragmatic surface was found to present a singular appearance. It had yielded to the pressure of the convex diaphragm, so as to present a concavity of great size, into which the right portion of the diaphragm accurately fitted. When the viscera were removed from the abdominal cavity, this portion of the muscle, distended and rendered convex by the thoracic effusion, presented a most striking contrast with the left, which was in its natural state. Some adhesions existed between the upper portion of the liver and the diaphragm.

The right pleura contained upwards of nine pints of an opaque, whey-coloured fluid, and was universally lined by a thick layer of flocculent lymph. The lung compressed, and, presenting wrinkled folds, lay against the mediastinum, its lower lobe somewhat projecting, and separated from the diaphragm by a large space. In its antero-superior portion was a cavity of the size of a walnut, filled with thick, brownish-yellow pus; this was covered externally by the pleura. On opening the pericardium we found its surface universally covered with lymph of a reddish colour, and formed into small, irregular masses or granules; but there was no adhesion. The whole surface was thus rendered exceedingly rough, particularly towards the apex, the situation in which, during life, the friction sound had been loudest, and most resembling the *bruit de râpe.* The lower portion of the ileum was in a state of great vascularity, and its mucous coat softened.

In the second case of this disease which I have recorded, we had an example of latent dry pericarditis supervening upon an old empyema of the left side, which had produced great displacement of the heart. In the present instance, however, we see the same disease following a recent pleuritic effusion of the right side, with extensive displacement of the liver. In both cases the disease was recognised, and the diagnosis verified by dissection, although none of the usual symptoms of pericarditis were present, and although the patients never complained of any uneasy sensation referred to the heart. In both, too, the diagnosis was founded on this principle, *the appearance of the phenomena of fremitus or rustling, as felt by the hand, with the stethoscopic signs as described, in a case in which, a very short time before, no such phenomena existed.*

In these two cases, although the pulse was regular, the action of the heart not altered in any new manner, pain absent, and the sound on percussion clear, yet a universal pericarditis was detected. I need scarcely remark that in this case our diagnosis was much strengthened by the observations on the former one. In one respect our diagnosis of these cases differed: in the former, the gradual cessation of the phenomena, except over the base of the heart, while the region of this organ continued clear on percussion, led us to conclude that a process of obliteration had taken place extensively; while in that before us, the persistence of the phenomena, both as to extent and intensity, enabled us to declare that no obliteration of any part of the cavity of the pericardium had taken place. The examination of the cases will show the correctness of the diagnosis in both instances.

Three circumstances are worthy of notice in this important case:—

1. The supervention of pericarditis in its last period.
2. Its occurrence in a heart under the influence of eccentric pressure.
3. The absence of all the usual symptoms of the disease, whether as regards pain or abnormal action of the heart.

In the case next to be given of the combination of empyema and pericarditis, the left pleura was the seat of the effusion.

CASE VI.—*Extensive Empyema of the left Pleura; Dexiocardia; acute latent Pericarditis; intense Friction Sound, disappearing with a nearly complete obliteration of the Pericardial Sac.*

A man named Lennon, aged 28, was brought to the Hospital early in January, 18—, labouring under the most aggravated dyspnœa. On examination I detected an extensive empyema of the left side, and the heart was observed to pulsate to the right of the sternum, but presented no morbid sound whatsoever. His symptoms had been at least of four months' standing, and he stated that he had observed the displacement of the heart a month previous to his admission.

On the 1st of February the patient came under my care, the displacement of the heart continuing, *but without the occurrence of any morbid sound in its pulsations.* He was treated by mild mercurials and narcotics. In the course of the week he began to suffer extremely from flatulent distention of the belly. On the 10th I made a careful examination of the whole chest. No change whatever was observed in the stethoscopic phenomena or impulse of the heart, but on the 12th, having placed my hand accidentally over the displaced heart, I was astonished at feeling a most distinct fremitus over its entire region, giving to the hand a sensation of two very rough surfaces rubbing violently one upon the other. On applying the stethoscope we found that the sound varied over different portions of the heart. At the base it was similar to the friction sound in ordinary cases of dry pleurisy, but towards the apex it closely resembled the *bruit de râpe* of Laennec, its point of greatest intensity being between the upper border of the third and lower of the fourth rib. We observed also that, if the stethoscope was moved to a distance of not more than an inch and a half from the situation of the heart, these remarkable phenomena ceased, though the contractions of the organ were heard distinctly. Pulse about 130, small, but not at all irregular; the sound of friction accompanied both sounds of the heart; dyspnœa very urgent, but the patient made no complaint whatever as connected with the heart. The cardiac region was freely leeched, and the patient ordered digitalis.

13th. The fremitus is remarkably diminished; the sound is analogous to the double *bruit de râpe;* heart's impulse less; no increase of dulness on percussion. From this period till the 17th the sensation and sound of rubbing gradually disappeared; it was only by close questioning that the patient admitted he had some pain at the right of the sternum.

On the 18th all fremitus and rasping sound had disappeared, except in a spot which could be covered by the stethoscope over the base of the heart and to the right side. In this situation a sound between *frottement* and a *bruit de râpe* was distinctly audible. The patient sunk on the 22nd.

Dissection.—The left pleura presented the usual appearances which occur in extensive and chronic empyema, its cavity contained nearly a gallon of sero-purulent fluid. The right pleura contained about a pint of perfectly clear serous fluid, and presented no effusion whatever of lymph on its surface. The pericardium appeared increased in size; it had lost its semi-transparency, and could not be made to glide over the heart. On opening its cavity, we found, with the exception of a small space at the base of the heart, exactly corresponding to the situation where the friction sound was last heard, that it was completely obliterated by recently effused lymph, which was reddish, and though soft, presented a considerable degree of consistence; so that when the two folds were separated by traction a vast number of laminæ, perpendicular to the surface of the heart, made their appearance. On the anterior portion of the ventricles, towards the apex, the union of the two surfaces was complete. Here the quantity of effused lymph was evidently much less than in the other parts of the cavity. Around the origins of the great vessels, particularly towards the right side, no union had taken place between the surfaces of the pericardium. Each face, however, was covered by lymph, presenting a considerable consistence, and giving the appearance which is produced when two smooth surfaces covered with a tenacious matter are suddenly separated.

This case I look on as one of extreme importance, as it was the first in which the positive diagnosis of an effusion of lymph on the surface of the pericardium was verified by dissection; and it must be recollected that the heart was extensively displaced by an empyema, and that the patient scarcely, if at all, referred any uneasy sensation to the situation of the recently suffering organ. The diagnosis was founded on the following circumstance, viz., the sudden appearance of the fremitus, and of the sound similar to the *bruit de râpe*, in a case which had been long under accurate observation, and which, two days previously, presented no such signs.

But in the progress of the case we added to our diagnosis, and I recorded it as my opinion that adhesion had taken place everywhere except over the base of the heart. This diagnosis was arrived at from observing the rapid subsidence of the signs under the influence of treatment, except in the above situation, *the region of the heart still continuing clear on percussion;* a proof that the disappearance of the signs was not owing to a liquid effusion; which opinion was still further rendered probable by the impulse of the heart continuing to be felt with the utmost distinctness.

The latency of pericarditis in both these instances would be by some attributed to the fact of its invasion during the last periods of life, but I do not believe that this explanation can be received, for I have witnessed the invasion and cure of pericarditis during the progress of an extremely chronic empyema of the left side occurring long before the death of the patient. Of this the following case is a good illustration:—

CASE VII.—*Chronic Empyema of the left Pleura; intercurrent latent Pericarditis affecting the displaced Heart.*

A woman, aged 26, after exposure to cold was attacked on the 10th of December, 18—, with symptoms of acute pleurisy of the left side. These had continued for nine days, when she was admitted into the Meath Hospital, with the usual symptoms and signs of extensive effusion into the pleura. The heart pulsated to the right of the sternum and in the epigastrium, but its sounds were natural. On the fourteenth day of her residence in the Hospital my friend, Dr. Thomas Brady, under whose special care the patient had been placed, discovered for the first time pericardial friction sounds in the displaced heart. It was stated to him by some of the pupils that these phenomena had existed for a few days previously. No new symptom attended this extension of disease; the pulse had not changed in character; it was 96, small and feeble; nor is there any notice of its ever having been irregular while the pericarditis continued. On the day when the sign was first observed the heart could be seen pulsating in the epigastrium; its sounds were audible over the anterior portion of the chest, but they had a peculiar muffled character, as if some soft body intervened and deadened them. Over the right side of the chest, and along the cartilages of the third, fourth, fifth, and sixth ribs, distinct double friction sound was audible, loudest at the line of the mamma, and persisting when respiration was suspended. These two sounds were followed by another, which was short and sharp; and the whole might be thus expressed: *pu-pu-pi.* No one symptom indicative of pericarditis existed.

Nine days elapsed, and the friction signs on the right side and across the sternum were even more distinct. Their intensity diminished, however, as the left side was approached, until they disappeared, leaving the sounds of the heart without friction, but still with the muffled character before noticed. Posteriorly the sounds of the heart were unaccompanied by any attrition sign, and percussion gave no evidence of pericardial effusion. No change was observed for seven days more, when it was found that the friction was scarcely perceptible; it could be detected at the cartilage of the fourth rib, and the sounds of the heart had lost the muffled character. From this period there was no return of pericarditic signs, although the patient lived for four months afterwards.

We have now reviewed three cases of the combination of empyema and pericarditis, and in them all we see a dry pericarditis, only revealed by physical signs. So latent, indeed, was the disease, that its existence would have never been suspected had not the employment of the stethoscope, in examining the progress of the pleuritic disease, led, as it were, accidentally to its detection. And yet, as has been before remarked, the heart was in all these cases under pressure. In two it was dislocated to the right side, and in one it must have suffered great pressure when we consider that the effusion actually displaced the liver.

The latency of the disease in these cases is to be explained by referring to the general law that the pre-existence of an important local or general disease seems to act in preventing the development of symptoms in the new affections that may be superadded. We cannot refer in these cases to the fact that the disease only supervened in the last periods of life; for, as we have seen in the last-mentioned example, the patient lived several months after the subsidence of the pericarditis.

Finally, the singular duration of the friction phenomena in the first case described demands our special notice. It is certain that they continued for sixteen days, and there is reason to believe that two or three days may be added to this period. I have never met with a case in which so long a time passed before organization of the false membrane took place, and the circumstance can only be explained by referring to the condition of the patient, who was all through suffering from aggravated symptoms of empyema, with copious expectoration and severe constitutional disturbance.

CASE VIII.—*Acute gangrenous Abscess of the Lung; Pericarditis.*

Of this combination I have observed a single instance. A man, aged 40, died after a fortnight's illness. His symptoms had been those of an acute pneumonic inflammation, and on admission his breath and expectoration revealed the existence of putrefactive action in the lung. The right side was generally dull on percussion, except at the root of the lung, where a cavity was detected by the usual signs. The pulse was feeble, and it is to be regretted that no careful examination was made of the heart. He died on the day of his admission. On dissection, the right lung was found in a state of purulent infiltration, and containing many small abscesses, some of which were quite superficial, and only covered by the pleura. The upper lobe was in the state of red hepatization. A cavity existed in the postero-inferior portion, the walls of which were gangrenous, and it contained a quantity of fœtid matter; many of the smaller abscesses were surrounded by a dark margin. The pericardium was everywhere covered with a coat of finely granular lymph. On applying the hand to the heart, the same sensation was produced as that from rubbing the tongue of a cow. The kidneys were in an advanced stage of Bright's disease.

Though we want a sufficiently extensive observation to warrant the conclusion that pericarditis, when combined with chronic disease of the lung, is generally latent, there appear strong grounds for such an opinion. Hence we might expect this latency in the combination of pericarditis with chronic tubercular disease, and the experience of Dr. Law goes strongly to confirm this view;[1] it is further probable that in a large proportion of the cases of complication with essential diseases, more or less of the character of latency will be observed. Hence, in cases of typhus fever, in the eruptive diseases, in diffuse inflammations, in erysipelas, and phlebitic and puerperal fevers, we may expect to meet with the character of latency, so far as symptoms are concerned, as much or even more than in rheumatic fever.

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[1] In a case of phthisis recorded by Louis, Recherches Anat. Path. sur la Phthisie, Obs. 19, pericarditis supervened during the last month of the patient's life. The pulse was frequent, unequal, irregular, and sometimes intermittent, and the impulse of the heart was increased. The invasion of the pericarditis, however, occurred simultaneously with that of a pleurisy of the right side; but there was no important symptom of the heart affection beyond the characters of the pulse. See also, Andral, Malades de Poitrine, Obs. 5.

See the Transactions of the Pathological Society of Dublin, January, 1841. Dr. Law exhibited a series of specimens of pericarditis, and in all those cases combination with chronic or acute disease of the lung existed. The pericarditis was principally detected by its tactile and acoustic signs.

We find the following illustrative case in the Clinique Medicale of Andral:—A lad, aged 17, was attacked with small-pox, which ran its usual course up to the seventh day, when, just as the pustules were in full maturation, the patient was attacked suddenly with dyspnœa. There was no cough nor bloody expectoration. During the eighth and ninth day the eruption remained stationary; then some of the pustules became black; others were filled with a reddish serosity, and between them livid petechiæ made their appearance. The dyspnœa increased, and death took place on the tenth day. A sero-purulent effusion into the pericardium, and a vivid injection of the great cul de sac of the stomach, were the only morbid appearances discovered.

COMBINATION OF PERICARDITIS WITH ANEURISM OF THE AORTA.

This combination appears to be rare, a circumstance the less remarkable when we bear in mind the infrequency of acute inflammation in aneurismal cases. Hence, the frequency of death by rupture into a serous sac. No case of the combination in question has occurred to myself, but the following example possesses some points of interest. It was communicated to the Pathological Society by Sir Philip Crampton, in 1845:

"A soldier, who had served in tropical climates, after having laboured under symptoms supposed to be those of pleuritis, was attacked suddenly with severe pain in the thorax. On examination Dr. Tice, the attending surgeon, discovered a pulsating tumour under the right mamma. After a few days he was seen by Sir Philip Crampton, who found a large pulsating tumour displacing the right pectoral muscle upwards and forwards. Its impulse was very strong, while that of the heart itself was feeble, and only one sound, believed to be the first, was audible. The diagnosis of aortic aneurism was made, and it was conjectured that a liquid effusion existed in the left pleura. On dissection a large false aneurism, with an opening into the vessel capable of admitting the thumb, was discovered. It sprung from the commencement of the second portion of the arch, and adhered anteriorly to the thoracic walls. From one of the ribs the periosteum had been removed by the action of the aneurism. The heart was not hypertrophied, but the pericardium was extensively inflamed, and the sac filled with fluid."

We here observe another instance of pericarditis arising in connexion with a chronic disease within the thorax, and with obscure or doubtful symptoms. The most interesting point, however, in the case, is the fact that in its advanced periods the heart gave but a single sound. Nobody can more fully admit the danger, I might almost say the impropriety, of discussing a recorded case while we assume that there has been an error in the observation; yet I cannot help believing that the single sound heard in this case was the second sound, and not the first, as stated in the communication to the Pathological Society, an opinion which the following circumstances seem to justify:—

1st. That I have never observed the extinction of the second sound of the heart in cases of aortic aneurism.

2ndly. I have noticed the weakening and almost complete extinction of the first sound in pericarditis; hence it becomes more than probable that the single sound heard in this case was the second sound, the extinction of the first being caused by weakness and semi-paralysis of the ventricles, producing in this way the physical signs which we observe in typhoid softening, or debility of the heart.

COMBINATION OF PERICARDITIS WITH TYPHUS FEVER.

When we recollect the rarity of secondary disease of the white tissues in typhus, we may anticipate that the occurrence of pericarditis under such circumstances is seldom met with; and, so far as inflammatory affections are concerned, the heart enjoys a singular exemption, as compared with other organs, while the system is under the poison of typhus. Thus, out of eighty-six cases recorded by Andral of death in severe fever, but thirteen exhibited any trace of alteration of the heart; and it is more than doubtful that the changes in these cases were of an inflammatory nature. I have myself only once observed the combination in question, but my recollection of the case is not sufficiently accurate to justify my giving it in detail. But while we admit the rarity of pericarditis in the typhus fever of this country, we know that in many affections having the typhoid chanot hypertrophied, but the pericardium was extensively inflamed, and the sac filled with fluid."

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TRAUMATIC PERICARDITIS.

There is no reason for believing that when the disease results from a direct injury, such as a blow or wound, the accident is attended by any special modification of physical signs. I have, however, seen a case in which the friction phenomena were developed in an unusual manner. A man received the contents of a gun, discharged at some distance from him, on the anterior portion of the left side. The gun had been loaded with small shot, and the pellets were scattered over a considerable surface, many of them not penetrating deeper than the skin. Most of these little wounds were received in the cardiac region, the integuments of which were dotted with small black spots, under many of which a grain of shot could be felt. The patient suffered principally from faintness and nervous depression; but that these symptoms were not the result of carditis was evident from the fact that they existed from the moment of his receiving the injury. For two or three days there was no indication of pericarditis; but after this time, and when the collapse and nervous depression had passed off, physical signs of a peculiar nature were developed over the region of the heart. There was no dulness on percussion, and the best idea of the signs may be given by stating that they consisted in the existence of many distinct points of intense friction sound, each of which, though extremely circumscribed, conveyed the impression of a resisting or cartilaginous deposit. These signs continued for several days, during which the friction phenomena subsided at certain points and appeared at others. There was no constitutional suffering, and but little, if any, local distress. The patient speedily recovered.

I think little doubt can be entertained that the pericardium was injured, while the inflammation, instead of spreading over the entire surface, was confined to the points of lesion. The character of the signs was such as I have never observed in idiopathic disease.

TREATMENT OF PERICARDITIS.

Although the principles of treatment of this disease are generally similar to those of pleurisy; yet it commonly happens that a more energetic practice is adopted in pericarditis than in inflammation of the pleura. From the importance of the organ engaged arises the apprehension of greater danger, and thus it often occurs that while the most active means are employed, the risk attendant on a too great weakening of the system at large, and also of the muscles of the heart, is overlooked. Such a line of treatment, especially as regards too free or repeated blood-lettings, is unnecessary, and generally dangerous.

In examining this subject we must separate the more violent cases of the primary disease, and perhaps also those instances where, in the course of a rheumatic fever, there is an explosion of pericarditis, from that larger class where the affection exists as one of a group of irritations, or as a mild though intercurrent disease. In such cases the boldness of treatment often betrays the timidity of the practitioner; he is terrified at discovering the disease, and his mind is more occupied with its name than its nature or actual condition. In this way great mischief is done, for the debility thus produced disposes the disease to change from the dry and comparatively innocuous form, to an unhealthy inflammation, attended with liquid effusion.

It is important, further, to observe, that although as above stated, the principles of treatment of the more violent forms are similar to those which guide us in acute sthenic pleurisy, yet the analogy only holds good up to a certain point, for it will be found that the period at which such treatment ceases to be advantageous or safe arrives much sooner in pericarditis than in pleurisy. In both diseases, it is true, we have to contend with a severe inflammation of a serous membrane, but in pericarditis a more important and complicated apparatus is engaged, giving rise to dangers foreign to the case of pleurisy. The period soon arrives when either from inflammation, paralysis, or the combination of both, the heart itself is weakened, and the patient is in danger of death from syncope, so that persistence in the reducing treatment may be followed by fatal results. The conclusion is obvious, that whatever may have been the necessity for depletion at the outset of the disease, we cannot press it in pericarditis to the same degree as in pleurisy.

In regulating our practice we derive great advantage from physical examination. So long as the impulse of the heart continues vigorous, its sounds remaining without signs of progressive diminution, and the patient's strength unimpaired, the dangers in question may be considered as remote; yet here it is not to be forgotten that the weakness of the heart, like that of the diaphragm and intercostals in pleurisy, *may supervene in a sudden manner.* In pleurisy such an accident is of comparatively slight importance, but in pericarditis it is one of great danger, threatening paralysis of an organ which is the fountain of life.

It is my conviction that the fatal result of some cases of pericarditis is mainly attributable to the perseverance, beyond the proper time, in the antiphlogistic treatment; the practitioner looking at the disease merely as a case of serous inflammation, and forgetting not only the results of irritation on muscular fibre, but the effect of great losses of blood in producing re-action.[1]

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[1] Dr. Hope strongly advocates the importance of energetic antiphlogistic treatment employed with the utmost promptitude:—

"The loss of a few hours at first may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. If the attack is recent, and the patient's strength will admit, blood should, in the first place, be drawn freely, and by a large incision, from the arm of the patient in the erect position, so as to bring him to the verge of syncope. From five and twenty to forty leeches, according to the strength, should then be applied to the præcordial region so soon as the faintness from the venesection disappears and re-action commences, which generally happens in the course of from ten minutes to an hour or two. Unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be repeated two, three, or more times, according to the strength, at intervals of from eight to twelve hours, or, what is a better rule, as soon as the pulse and action of the heart denote recommencement of re-action.

"It is not, however, in every case that so active a treatment is required. I have seen a single prompt and abundant application of leeches, or a cupping, at once subdue every formidable symptom. When the patient, either from age, a feeble constitution, or the advanced state of the malady, cannot bear extensive depletion, local bleeding is, according to my observation, decidedly preferable to general; but it should be practised effectually, by cupping to twenty ounces or more, or by the application of from twenty-five to thirty or forty leeches. When, from depletion having already been carried to a great extent, or from the ad-

Let us now suppose that we have a case of uncomplicated pericarditis in its earlier stages, and occurring in a patient whose strength is but little impaired: in such a case a single bleeding from the arm appears, on the whole, justifiable, but its repetition will be a matter for careful consideration. Under these circumstances we must examine the force of the heart, not only as indicated by the pulse at the wrist, but by the actual strength of the impulse, and the character of the first sound especially. If the impulse continues vigorous, and the first sound undiminished, we may be less apprehensive of the use of the lancet. On the other hand, if, after depletion, the impulse has manifestly declined in

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vanced stage of the disease, it is not safe to draw much more blood, yet it appears expedient, from the persistence of pain, &c., to draw some, I have generally found that a smaller quantity drawn by cupping, produced more effect than a larger by leeching. The cause of this probably is, that by cupping it is drawn more expeditiously.

"I may finally remark that, though blood ought to be drawn with all the vigour that I have described when the usual indications for its emission exist, yet, in cases where mercury is employed, as presently to be described, those indications so soon cease, from the controlling power of this remedy, *that the total quantity of blood lost will rarely be considerable.*"

He adds:—"I feel satisfied that a degree of activity, in the first instance, which to some may appear excessive, is an ultimate source of economy to the strength of the patient, for the disease is subdued at once, and the protracted continuance of depletory measures, the most exhausting to the constitution, is rendered unnecessary."—*Op. Cit.*

But, without denying that in some cases such a course as is here indicated may be proper, we must not forget the effect which this advice may have on some of our brethren, whose minds are not sufficiently purged of the erroneous doctrines of inflammation, so long the opprobria of our Schools of Medicine and Surgery. There are many who could not, like Dr. Hope, discriminate between cases requiring such a vigorous treatment and those of a very different kind,—who know the disease only by name, and are unaware that the former are the exceptional cases. On this subject Dr. Wood has some excellent remarks. After observing that the heart is often stimulated by great losses of blood, he says:—"These are not arguments against blood-letting, but only against its abuse. The application of the remedy is to be guided here exactly on the same principles as in other cases of serous inflammations. The stimulating quality of the blood should be reduced by depletion, and the direct sedative effects of its loss upon the heart be obtained without pushing it to the point calculated to produce re-action. The theory which urges to any risk in order to avoid the terrors of adhesion should not be allowed to have any weight." See his Treatise on the Practice of Medicine, Philadelphia, 1849; Art. Pericarditis; also Dr. Todd's work on Gout and Rheumatism, 1843, p. 197.

force, while the first sound is lessened, great caution must be used before we repeat the general bleeding.

The force of the contractions of the heart, as indicative of the safety of further bleeding, is only valuable when no intervals of weakened action have occurred. Where it has been an unchanging condition, and especially when the heart's action is regular, or nearly so, we may, of course in addition to other circumstances relating to time, and the age and strength of the patient, adopt it as an indication that another bleeding may be performed without risk. But our great reliance is to be placed on local bleeding, and the best mode appears to be the employment of leeches, in relays, beginning with twenty or thirty, and gradually reducing the number on each application. Two or three applications may be made in the twenty-four hours, a warm poultice being employed during the intervals. At the same time it will be advisable to induce a mercurial action by such means as are within our reach, and it is probable that the plan of giving a full dose of calomel,—say from ten to twenty grains,—at long intervals, as recommended by Dr. Graves, will best answer our expectations. "If," says Dr. Graves, "a person is seized with very acute pericarditis, how unavailing will be our best-directed efforts unless they be succeeded by a speedy mercurialization of the system! In proof of this assertion I might adduce a considerable number of cases of pericarditis, treated both in hospital and private practice, and might triumphantly compare the results with those obtained in the continental hospitals, as recorded by some of the most eminent German and French physicians. When even the most violent attacks of pericarditis, are met with copious venesections, repeated leeching, and the rapid injection of calomel, few patients will be lost. If, on the contrary, the practitioner relies solely on the lancet; if, in the beginning, as I have seen done, he applies a blister over the heart, and if he defers the exhibition of calomel, or *insufficiently uses it*, then will he have occasion to regret the consequences, and witness either the speedy death of his patient, or his condemnation to the sufferings entailed on him by adhesions, valvular disease, and the other sequelæ of badly treated pericarditis."[1]

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[1] Clinical Medicine, page 803.

This method of using calomel is that advocated by Dr. Johnson in the treatment of the diseases of tropical climates, and consists in the exhibition of scruple doses once or twice daily. The patient must take no cold fluids, acids, or fruits, but should drink freely of warm barley-water. By this treatment it is found that mercurialization may be effected without producing any considerable amount of abdominal distress; and it is remarkable that the action of the medicine is attended with an abatement of fever, and a decided diminution in the frequency of the pulse. Dr. Graves further states, that by using the remedy in this way he has cured sixteen patients, without any permanent injury to the constitution. Finally, he observes, that when, in cases neglected at their commencement, the diminution of fever and retardation of pulse does not follow the mercurialization of the system, it is a bad sign; still worse is it if the fever increases, for he believes, and in this opinion I agree with him, that this is owing to an aggravation of the disease, and not, as is often supposed, to the action of the remedy.

In the second stage of the disease our principal reliance must be on blisters; but we may apply leeches again and again on any new excitement of the heart. At a more advanced period, when immediate danger is not to be apprehended, and that liquid effusion exists, we shall probably obtain advantage from the repeated application of tincture of iodine over the pericardial region; but this suggestion is made more from our favourable opinion of this remedy in pleurisy, than from any actual knowledge of its effect in pericarditis.

As to the use of digitalis in this disease, so long as fever exists, and the heart remains in the state of inflammatory excitement, the remedy seems inefficacious. Again, in the more advanced stages, and when the organ has been weakened, its exhibition might be dangerous. There is a period, however, in which we may employ the medicine, namely, when, after all fever and physical signs of pericarditis have subsided, the heart acts with undue force, a condition sometimes attended with valvular murmur, but in other cases without it. In this latter instance especially, we find advantage from small and repeated doses of digitalis. Should the medicine disagree, the hydrocyanic acid may be substituted.

itself. Many of the articulations were swollen and painful, and she also had pain in the heart, palpitation, and a great amount of dyspnœa. Percussion showed increase of dulness over the heart, while an intense friction sound could be heard from the entire surface of the organ, audible also over the whole anterior portion of the chest, and in the left side posteriorly. The action of the heart was violent, yet the pulsations did not resemble those of hypertrophy. Pulse 108, jerking. The carotids had a strong and visible pulsation. She was treated by a single bleeding, followed by leeches to the cardiac region, while calomel and opium were exhibited. Symptoms of great debility soon appeared, while the friction sounds continued intense, and the præcordial distress was but little abated. Under these circumstances she was ordered to have a small number of leeches applied over the heart, while at the same time we gave four ounces of wine. Next day there was a distinct improvement in the general and local symptoms; the wine was continued, and it really seemed to act as a sedative on the inflamed heart. In a few days the friction sounds wholly disappeared, and her recovery was most satisfactory.

This case is strongly illustrative of the efficacy of wine in certain conditions of pericarditis, and it is important to observe, that although on both occasions of the administration of stimulants the general state of the patient was that of great debility, yet there was no evidence of failure of the heart's action, which was excited and vigorous. Thus we find that there are at least two cases of pericardial inflammation in which wine may be employed: one, that of uncomplicated disease, where the muscular action of the heart is failing; the other, a case of secondary, or at least complicated pericarditis, with general debility and a typhoid state, although no signs of cardiac weakness or paralysis have so far appeared. Under such circumstances, then, *even a vigorous action of the heart, a jerking pulse, and an increased action of the carotids, do not necessarily contra-indicate the use of wine;* nor should the existence of the recent valvular murmurs of endocarditis in such cases debar us from the use of the remedy. For we often meet with the same general conditions now described, yet without any affection of the serous covering, while the endocardium is engaged, yet in which wine proves of the greatest service.

prevent the use of wine, if circumstances require it. In two cases especially, namely, cerebritis and pericarditis, we find the greatest timidity in practice with respect to the use of wine. Yet, even in the first case it may be required, and in the second its employment is imperative, when, as too often happens, excessive depletion has been resorted to. Again, if the signs of muscular weakness, such as we have indicated, have appeared; if there be evidence that the heart, previous to the attack, was in a weakened state; and lastly, when a collapsed or typhoid condition of the system exists, we must give wine, quite irrespective of the physical condition of the heart. This may be done safely, and with great advantage. In the following case wine was employed with the best effects.

CASE IX.—*Two attacks of Rheumatic Carditis, within a period of seven months, with an intervening seizure of apparently nervous palpitation; use of wine; recovery.*

A young woman was admitted into my wards in December, 1850, labouring under acute arthritis. She was greatly prostrated, and suffered much from the affection of the joints. Pressure over the heart caused some uneasiness; but this symptom, and a slight prolongation of the first sound, were the only indications of disease. In a few days friction sound was audible over the base of the heart. The prostration had increased. The treatment which had been adopted was the use of mild mercurials with opium, and the application of small numbers of leeches to the joints. On the day on which we discovered the pericarditis, wine was ordered, at first with caution, but subsequently with greater freedom, and with the best results. The patient improved daily, so that in a short time no friction sound could be detected, unless when strong pressure was made over the heart. She was finally dismissed in good health, but with a feeble murmur heard at the apex of the heart. Four months having elapsed, she was re-admitted, labouring under a nervous attack, which had set in with delirium, and was attended with excited action of the heart, but no sign of carditis could be discovered. This illness subsided in a few days, and she left the hospital. In August, however, she returned; she had been exposed to wet and cold, and rheumatic fever again showed itself. Many of the articulations were swollen and painful, and she also had pain in the heart, palpitation, and a great amount of dyspnœa. Percussion showed increase of dulness over the heart, while an intense friction sound could be heard from the entire surface of the organ, audible also over the whole anterior portion of the chest, and in the left side posteriorly. The action of the heart was violent, yet the pulsations did not resemble those of hypertrophy. Pulse 108, jerking. The carotids had a strong and visible pulsation. She was treated by a single bleeding, followed by leeches to the cardiac region, while calomel and opium were exhibited. Symptoms of great debility soon appeared, while the friction sounds continued intense, and the præcordial distress was but little abated. Under these circumstances she was ordered to have a small number of leeches applied over the heart, while at the same time we gave four ounces of wine. Next day there was a distinct improvement in the general and local symptoms; the wine was continued, and it really seemed to act as a sedative on the inflamed heart. In a few days the friction sounds wholly disappeared, and her recovery was most satisfactory.

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If we consider that extensive series of cases in which pericarditis occurs, either as secondary to a general or essential disease, or as one of a group of local inflammations, we shall find many cases in which wine may be used with liberality, even though endocarditis be present. Excluding the complication with ordinary rheumatic fever, we have to deal with pericarditis in connexion with the diffuse inflammations, or the low erysipelatous state; and again, in the pyogenic condition, as in the remarkable cases described by Dr. E. M'Dowel;[1] in typhoid pneumonia; and in the complication with delirium tremens from excess, already alluded to, which is so often attended with a typhus or typhoid fever. Many other cases might be specified, but enough has been said on the general question. There are two cases, however, sufficiently common to deserve notice here; one is the occurrence of the disease in the broken down, gouty constitution, and the other that in which pericarditis attacks a heart in the earlier stages of fatty degeneration. Here the greatest faults in practice, both of commission and omission, are often seen; the original disease is unsuspected, and the patient held to have been in good health up to the time of the appearance of carditis, when the lancet on the one hand, and the debarring of stimulants on the other, at once reveal his condition, in most cases when it is too late to mend it. In truth, it may be said that no man is fit to treat general disease or local inflammation, especially its secondary forms, until he has conquered that fear of stimulants which a long course of erroneous teaching has instilled into his mind.

When the disease is only indicated by the signs of dry pericarditis, without fever or excitement of the heart, little more is necessary than the moderate use of local depletion; but the slightest appearance of excitement of the organ, even though unattended by any new sign of exocardial or endocardial disease, should be at once met by an application of leeches, followed by poulticing; in fact, the cardiac disease is to be treated precisely as that of the joints. I have seldom used mercury in rheumatic pericarditis, where the symptoms were mild or wanting, and the pulse regular; and it does not appear that the mere fact of complication with dry pericarditis should lead to any special alteration in our treatment of rheumatic fever. Great advantage will be obtained from the use of poultices; they are particularly applicable in this form, for the patient can bear their weight without the suffering which they occasion in the more violent and idiopathic disease.

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[1] See his Observations on Periostitis and Synovitis, Dublin Journal of Medical Science, First Series, vol. iv., 1834.

Upon the merits of specific treatment in gouty or rheumatic pericarditis, I can say little from my own experience, for I have always been reluctant to adopt such a course. When either fever or cardiac excitement exists, colchicum and bark should be used with extreme caution, but the use of opium in free doses is not so objectionable. Where great pain attends the disease, or that the affection simulates angina pectoris, Dr. Latham strongly advocates the use of opium.[1]

Finally, it may happen not only in the secondary but the primary forms of this disease, that after the first violence of the attack has been subdued, an effusion of liquid, more or less copious, remains in the pericardium, and a condition is produced, analogous to that of chronic empyema following an acute pleurisy. In such a case we may employ mild mercurials, followed by the internal and external use of the preparations of iodine, while the action of the absorbents is assisted by the use of blisters or other counter-irritants. In such a condition the operation of tapping the pericardium, suggested by Senac and practised by Desault, and in recent times by Schuh, may be found advisable. I have no experience of this operation, yet although the difficulties and risks attendant on it are probably greater than in empyema, we cannot but hope that the puncture of the pericardium will, like that of the pleura, be soon deprived of much of its danger and difficulty.[2]

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[1] See Lectures on Clinical Medicine, &c., vol. i.

[2] In one case operated on by Dr. Schuh, of Vienna, the symptoms of hydrops-pericardii were so severe as to threaten suffocation. A trochar was introduced between the third and fourth ribs, very near to the edge of the sternum, and between it and the course of the internal mammary artery. At first only a few drops of blood flowed out; a small bougie, passed along the canula, touched the great vessels, the pulsations of which were distinctly felt. The operation was

Treatment of Rheumatic Pericarditis.

When the true relation between rheumatic fever and the different forms of carditis is considered, it will appear that the activity of treatment necessary in idiopathic pericarditis is not likely to be called for in the rheumatic variety. Whether the doctrine of Bouillaud, that the heart in arthritis is to be looked on as an additional articulation, be or be not adopted, we may hold that its irritations are subject to the laws which govern the affections of the joints. Like the articulations, we find it liable to every shade and variety of irritation, from the slightest to the most severe. Like them, too, we see it exhibiting great inconstancy in the mode of succession of the different morbid processes which attend its diseased state; and lastly, like many of the individual joints in rheumatic fever, we may see it completely exempted from any attack; nor can we say why this is so; why it is that in one instance the heart escapes, and in another is attacked; nor why its irritations in some cases precede, in others follow, or again, occur simultaneously with the inflammation of the joints.

Rheumatic pericarditis is, then, essentially one of the class of secondary local diseases, and to its treatment we must apply those maxims which guide us in the management of all such affections. The importance and, indeed, the absolute necessity, of making a daily examination of the heart while we are engaged in the treatment of a case of rheumatic fever has been insisted on by several

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immediately repeated between the fourth and fifth ribs, when there flowed out slowly, and in a stream, a certain quantity of reddish serosity, (see Medico-Chirurgical Review, vol. xxxvii., p. 587.) It is stated that relief followed the operation, and that at the end of the third week the effusion into the pericardium had disappeared. I am unable to ascertain whether this was a case of partial dropsy of the pericardium or of effusion into the sac, resulting from pericarditis. The case has but little value except with reference to the place of puncture. Two cases are given by Dr. Karnwagen, of Cronstadt, in which immediate relief followed the operation, and in one, a permanent cure. In the latter case not less than three and a half pints of fluid were drawn off, and during the operation air entered the cavity of the pericardium. In five months the patient might be considered convalescent. (See British and Foreign Medical Review, vol. xii., p. 250.) But neither of these cases appear satisfactory, and the length of time between the operation and the final recovery is remarkable, if we assume that an effusion had been removed by tapping. The diagnosis between mere dropsy of the pleura and the pericardium is not always free from difficulty.

writers; but it cannot be too strongly impressed on the mind of the practitioner that, valuable as the discovery of the signs of an inflamed pericardium may be, it is not for these alone that he is to look, but rather for the indications of excitement of the heart, whether attended or not by the signs of exocardial or endocardial disease. In other words, the sudden appearance, or the previous and continued existence of increased action of the heart should lead him not only to anticipate an attack of pericarditis, but should make him adopt the precautionary measure of local depletion, even though no friction sound or valvular murmur whatever be present.

But further, it may be laid down that any abnormal or unusual condition of the heart should awaken our suspicions, pending a case of rheumatic fever or general rheumatic disease. The following conditions may be specified among others:—

1. Excitement of the heart's impulse, without any corresponding state of the pulse, unattended by endocardial or exocardial murmurs.

2. Excitement of the heart and pulse, attended with a ringing sound of the ventricular contraction, appearing for the first time.

3. Sudden depression of the heart's action in force or rapidity. The first character may not be revealed by the pulse.

4. Sudden irregularity, without any other morbid sign.

5. Doubling of one of the sounds of the heart. This is not uncommon; it is much more frequent with respect to the second sound, and I have observed it to disappear on the patient assuming the erect position.

6. Prolongation of the first sound. This sign appears to depend, not on any valvular affection, but on some altered condition of muscular contraction.

It is hardly necessary to state that these conditions are not always followed by well-developed symptoms or signs of pericarditis or endocarditis, but that they indicate a manifest proclivity to disease is certain, and we find them arising in a state of system in which cardiac disease is of common occurrence. We find them often followed by the ordinary physical signs of the affection; and lastly, they are removable by local antiphlogistic treatment.

In practice, we may adopt the following arrangement of the cases in which manifest physical signs appear:—

1. Dry pericarditis, without excitement of the heart or valvular murmur.

2. Dry pericarditis, with excitement of the heart, but yet without valvular murmur.

3. Dry pericarditis, with excitement of the heart, and attended with valvular murmur.

4. Pericarditis with excitement of the heart, attended by valvular murmur, and the signs of a progressive liquid effusion.

The order in which these cases are arranged will mark their relative importance, and the degree of activity of treatment which they will require.

Whether rheumatic pericarditis demands any special modification of treatment is still an open question. The degree of activity of interference with the disease will, of course, depend not only on the character of the attack, but on the period of the fever in which it arises, and the strength and actual condition of the patient. In the last two forms it will be generally right to use mercury, pushed to salivation, not only with the view of controlling the pericarditis, but with the hope of preventing a chronic disease of the valves. Opium is generally useful, but I have never found that colchicum had any beneficial effect either in pericarditis or rheumatic arthritis, while the inflammatory fever continued.

APPENDIX TO THE PRECEDING SECTION.

I. *Physical Signs.*—Among the rarer forms of these phenomena is to be noticed the clicking sound described by Dr. Walshe. He says: "Occasionally sounds are heard of peculiar clicking character (only one or two with each beat of the heart,) which are only distinguishable at the time from modifications of the valvular sounds by their non-synchronism with these, and by the extreme irregularity of their occurrence. I have satisfactorily traced these clicks to the pericardium, and further, in all probability, to the separation (without attrition) of surfaces glued together with exudation matter."[1] Dr. Walshe further observes that he has never detected this clicking sound, except in the site of the large vessels.

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[1] Practical Treatise on Diseases of the Lungs and Heart, and of other Organs, 1851, p. 230.

This sign is not of common occurrence; its irregularity, and want of correspondence with the valvular sounds, are sufficient to prevent our mistaking it for that doubling of one of the sounds of the heart (generally the second) which has been noticed in the preceding pages.

II. *Effects of Adhesion of the Pericardium upon the Heart.*—At the time when I made a communication to the Pathological Society of Dublin on this subject, and also when my observations on it in the present work were written, I did not know that the views which I ventured to put forward had already been adopted and published by two distinguished writers, Dr. Barlow and Dr. Chevers. In the Gulstonial Lecture for 1843, Dr. Barlow not only states that hypertrophy and dilatation do not of necessity follow on obliteration of the sac, but that the latter condition in most cases tends to produce atrophy of the heart.[1] Dr. Chevers' papers will be found in the ninth volume of Guy's Hospital Reports. Dr. Walshe also observes that an atrophied state of the heart appears sometimes to follow from the formation of false membrane on its surface.[2]

The latest writer on the subject of adherent pericardium is M. Forget. He believes not only that adhesions of the pericardium must be considered as a serious pathological condition, and one calculated to interfere with the heart's action, but that its existence can be determined by the careful consideration of the history and actual phenomena of the case. He specifies (1) a tumultuous and confined action of the heart, consequent on the ordinary signs of pericarditis, or existing with other diseases, which do not explain the disturbance of the circulation. (2) The smallness, inequality, and irregularity of the pulse, indicating the difficulty experienced by the heart in performing a complete contraction. (3) The præcordial anxiety, dyspnœa, and tendency to fainting, derivable from the preceding causes. (4) The usual consequences of obstructed circulation, such as œdema, cyanosis, &c. A general adhesion, according to him, may be diagnosticated, when, after the subsidence of the friction sound of pericarditis, the heart assumes a permanently tumultuous and irregular action.

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[1] Medical Gazette, 1847.

[2] Op. Cit., p. 452.

I do not think that M. Forget has added much to our knowledge of this subject. A reference to the propositions attached to my original memoir will show that the diagnosis of adhesion, from studying the friction phenomena of the heart, was made long ago. It is certain that in some cases of pericarditis a tranquil state of the heart follows the organization of the lymph, while, in others, a permanently irregular action may be established. But M. Forget has failed to show that this irregular and tumultuous action is attributable to adhesion, for all the symptoms indicated by him may occur independently of any preceding pericardial disease. Again, after an attack of inflammation of so complex an organ as the heart, there may be other causes for disturbance of its action. The heart may be weakened; it may be in the first stage of irritative hypertrophy; coagula may have formed in its cavities; or a chronic endocarditis be in progress.

M. Forget depends on the coincidence of cessation of friction sound, with increase of disturbance of the heart's action, as the chief ground of diagnosis of adherent pericardium; and he observes that the pulsations of the heart which depend on valvular disease are more defined and less tumultuous, and are almost always accompanied with bellows murmur, constituting the pathognomonic signs of this condition, while that of adhesion is precisely the absence of this sound. His statement, then, comes to this, that if, after an attack of pericarditis, with friction phenomena, which latter have disappeared, the heart's action is tumultuous, the absence of valvular murmur should lead to the diagnosis of an adherent pericardium.[1]

From what has been now said the conclusion presses upon us, that while on the one hand we may have an obliterated, or nearly obliterated pericardium, without any of the conditions of the heart indicated by M. Forget, so, on the other, the signs which he has given are only conclusive, so far as the disappearance of the friction phenomena are concerned.

Finally, the researches of Dr. Gairdner have led him to the conclusion that as the adherent pericardium, at first uncomplicated, may in certain cases result in extreme hypertrophy of the heart, yet that in other cases it may not only fail to produce this effect, but appear altogether powerless in opposing the atrophy of the heart resulting from chronic disease.[1]

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[1] Précis Théorique et Pratique des Maladiés du Cœur. Par L. Forget, Strasbourg, 1851.

ENDOCARDITIS.

The term endocarditis has been but recently introduced into medicine, as designating the acute or chronic inflammation of the lining membrane of the cavities of the heart, and more especially its valvular apparatus. As in the case of gastro-enteritis, we find that both the term and the description of the disease belong to the physiological school, which refers so many affections to a simple inflammatory origin. But to every one who has studied the history of medicine for the last half century, it is obvious that the doctrines of that school were pushed too far, and that experience has shown not only that we are unable to refer fevers and many abdominal diseases to a gastro-enteritis, but that we cannot attribute all the organic diseases of the valves to inflammation of the endocardium. Still it is not to be denied that, for our knowledge of the great phenomena of gastro-enteritis and endocarditis, we owe every thing to the physiological school, and, in gratitude for the benefits it has conferred on medicine, we may well excuse its disciples for having overstepped the limits of strict induction.

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[1] Dr. Gairdner observes that "the only view which seems to harmonize these conditions is the supposition that the free motion of the heart within the pericardium is required in health, not so much to meet the necessities of the circulation in its tranquil and ordinary condition, as to provide for the contingency of excited action, and to give abundant scope for the smooth and painless motion of the heart under those circumstances in which the habitual equilibrium of the circulation is disturbed. Such circumstances are of daily occurrence; in the healthy and vigorous, from superabundant use of bodily exertion; in the sick and debilitated, from the more sparing use of it; in all, but especially in the nervous and excitable, from mental emotion, and a variety of minor causes. These temporary excitements are, however, to a great extent controllable; and on this fact depends, I believe, the practical application of these principles to the management of adherent pericardium, where it is known or suspected to exist."—*On the favourable Terminations of Pericarditis, and especially in Adhesion of the Pericardium, with cases illustrating its Secondary Effects on the Heart*, by W. J. Gairdner, M. D. Edinburgh Monthly Journal of Medical Science, 1851.

If, excluding the possible results of these lesions, we limit our consideration simply to their earlier periods, in which the pathological characters of acute irritation are developed, we shall at once perceive the leading practical error of the followers of Broussais, namely, that they took as their sole guide, the visible, tangible evidences of pathological anatomy, and did not recognise that the same anatomical changes might occur in essentially different states of the system, and have opposite relations to the constitutional condition; in one case the local disease being the cause of the general disturbance; in the other, truly its effect, though, when developed, capable of a re-action on the economy.

Pathological science has shown that organic changes may spring from an infinite variety of sources, and though with reference to the diseases of the white structures, inflammation appears to be one of the causes most frequently met with, yet we cannot, in the present state of our knowledge, reduce all valvular diseases of the heart to the formula of an acute or a chronic inflammation, and it will be better, practically, to consider chronic valvular disease as an affection *sui generis*, into the treatment of which the question of existing inflammation does not necessarily enter; and to place under the head of endocarditis only those cases in which, with co-existing signs of local irritation and general systemic disturbance, the signs of valvular lesion are more or less quickly established.

Endocarditis may be observed as a primary idiopathic affection; as a secondary lesion in various constitutional maladies; as a simple disease, or occurring in connexion with inflammation of the other cardiac structures; and lastly, it may be associated with analogous disease of other and even remote organs. The general formula for its detection is the occurrence of symptoms of cardiac irritation, followed or accompanied by signs of valvular lesion. If signs of pericarditis are present, the diagnosis will be of endo-pericarditis; if they are absent, of the simpler form of the disease. In the complicated cases it may precede, follow, or accompany the peripheral inflammation, and it may arise in its most acute form in a heart whose valves are already far advanced in disease.

So imperfect is our knowledge, that we cannot say how far the symptoms of general endocarditis differ from those of a partial affection; whether the disease in the right cavities presents phenomena different from those of inflammation of the left auricle and ventricle; nor can we, if we exclude the signs of valvular obstruction or erosion, declare whether the disease is attended with any proper physical signs. For the polypoid concretions, the false membranes spread over the cavities of the heart, the fissures, fungosities, and other alterations of the endocardium, are as yet incapable of being diagnosticated, their vital and physical effects merging into the general group of phenomena which attend diseases of the heart.

It is, then, by ascertaining the recent production of a valvular lesion we discover an endocarditis, and even this may not be always conclusive, for we shall see that diseases of the valves, whose origin is at least doubtful, are yet capable of a rapid and almost sudden development.

In practice, however, the disease may be considered in the following forms, which are given in the order of their frequency. First, it may accompany, follow, or precede an attack of pericarditis. Secondly, as occurring without pericarditis, when it is in general manifested by symptoms of cardiac irritation, with signs of recently-formed valvular disease. The absence of pericarditic signs may be owing to the actual want of any peripheral irritation, or to the obliteration of the sac by previous disease. Thirdly, we find that, without any symptom which would lead to the suspicion that the heart was diseased, endocarditis may be insidiously and silently developed in the course of rheumatic fever. Fourthly, symptoms of irritation of the heart may occur in a case where the organ has been previously diseased. These may be shown either by an increase in the violence of the old, or in the production of new symptoms; or lastly, by making manifest the signs of former organic disease, which up to the period in question had been unrecognised or wanting. Fifthly, symptoms of cardiac irritation may be developed, unattended by any evidence of valvular lesion. This form is of rare occurrence, and I put it forward with diffidence; but I have seen cases which could not be explained upon any hypothesis, except that of the absence of murmur in endocarditis.

These considerations apply only to the acute forms of the disease; for the diagnosis of chronic endocarditis, especially where we have not had an opportunity of studying its early stages, is difficult, and the differential diagnosis between it and valvular affections of another nature seems to be, in the present state of our knowledge, quite impossible. Even where the origin of the disease has been inflammatory, a great practical evil may arise from our continuing to view the case as one of chronic endocarditis, for experience teaches that in many of such instances a tonic and stimulating treatment will be attended with much happier results than can be obtained by the antiphlogistic system.

Endocarditis being most frequently met with at the left side of the heart, and its physical signs being developed principally at the orifices, it follows that the chief grounds for its diagnosis will be the recent production of mitral or aortic valvular murmur, in cases where the local and general phenomena are indicative of cardiac irritation.

To explain why it is that not only the physical signs of endocarditis, but also its more obvious pathological changes, are confined to the valves, is difficult. The recent production of a valvular murmur under general and local irritation may be held to imply some mechanical change in the valve itself, and we know that almost all the alterations or irregularities of the latter are competent to produce murmur. The very early appearance of this sign in acute endocarditis leads to the inquiry whether there be other causes for the alteration of the valve besides its inflammatory thickening, or the deposition of lymph on its surface. It is not improbable that those bundles of muscular fibres which govern the action of the valves, either participate in, or suffer from the endocardial inflammation, and, as a result, that whether their contractile force was augmented, as in spasm, or weakened, as in inflammatory paralysis, the valve would be thrown into a new and unnatural condition, and a murmur be developed even before its disorganization had taken place.[1]

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[1] I have specified paralysis as a possible condition, but in connexion with the early development of murmur there is greater likelihood that more value is to be attached to the opposite state. We may here refer to what has been already noticed in the typhoid softening of the heart, in which there is nothing more remarkable than the absence of valvular murmur. See Dublin Journal of Medical Science, First Series, vol. xiv.

If, however, dismissing these considerations, we admit that the valves are more prone to inflammation than the membrane lining the cavities, we are forced to inquire, what are the circumstances which cause this difference. The structure of the valves, so far as we know, does not differ from that of the endocardium generally considered. This at least is true of the auriculo-ventricular valves; but when we consider the anatomical relations of the membrane, we find that the endocardium of the cavities is in contact with the red muscular tissue, while that of the valves is a free serous structure. This, while it would not explain the greater liability to disease of the valves, might throw some light on the frequency of their chronic disorganizations.

It may be inquired whether those portions of the endocardium in contact with red structure have, from that very circumstance, any power of resisting inflammatory action, which is denied to the valves; or again, whether, from a superior vitality, they are endowed with greater energy, so as to rapidly organize and convert into transparent structure such exudations as may be formed upon them. When we come to speak of the markings of the ribs on the pleura, after pleuro-pneumony, we shall find that, while the serous membrane under the intercostal spaces may be, and often is, in a state of transparency, those portions which correspond to the ribs are opaque. In several cases I have found this opacity to depend on the existence of adipose structure, proving that the processes of transformation of lymph were different, according as the membrane was in contact with muscular structure, on the one hand, or with fibrous tissue, on the other. Should this analogical view possess any value, it may explain why the transformation into cartilaginous, bony, or atheromatous matter, is so often seen in the valves, and so rarely in the lining endocardium.

As we have ventured into the field of speculation, one more inquiry or suggestion may be made. Is the greater liability of the valves to inflammatory disease, in any degree connected with their relation to the tendinous filaments of the papillary muscles, which, in a case of rheumatic fever, at least, may be supposed to be more liable to disease than the remaining internal structures of the heart?

The symptoms of endocarditis are not yet fully ascertained or defined, and it is doubtful whether its diagnosis will ever be established with the same accuracy as that of pericarditis. Many circumstances occur to make this diagnosis difficult. Of these we may specify, first, the rarity of the disease in an uncomplicated form; secondly, the frequent co-existence of pericarditis; and thirdly, the general similarity of its constitutional symptoms with those of the latter disease. In truth, we rarely meet with a case of simple idiopathic endocarditis fit to be considered as a type of the signs and symptoms of the disease. Such a case at least has never occurred to me.

But yet we can often determine the existence of this affection, always provided that, with a careful study of the history and symptoms of the case, we combine the results of physical examination, for so closely do the symptoms of pericarditis and endocarditis resemble one another, that it is only by auscultation and percussion that, in many cases at least, we can hope to distinguish them.

Like pericarditis, this affection is often latent, causing little or no distress to the patient, no irregularity of the heart, nor any other symptom of irritation. This frequently occurs in rheumatic fever, and the practitioner is often surprised by his patient showing symptoms of valvular disease after an apparently perfect recovery from the fever. Latent endocarditis may thus exist, and the disease be only recognised when it is no longer curable.

Dr. Hope is of opinion that endocarditis more frequently exists without pericarditis, than pericarditis without endocarditis. I have come to a different conclusion. Doubtless, if we were to set down all the cases of organic valvular murmur, even of a somewhat recent date, as examples of endocarditis, we should have abundant instances of the apparently isolated disease. But when we remember how commonly pericarditis is latent—so latent that it scarcely disturbs the action of the heart—we should be reluctant to set down as simple endocarditis those cases in which a pericarditis has never been recognised, especially when we recollect that there are other causes for valvular disease besides inflammation; and, on the other hand, it is to be noted that the occurrence of acute pericarditis, without any present or subsequent valvular murmur, is sufficiently familiar to the clinical observer. In the present state of my opinion on this point I would place the cases in the following order of frequency:—

1. Acute pericarditis with endocarditis.
2. Acute pericarditis without endocarditis.
3. Endocarditis without pericarditis.

In the cases where more prominent symptoms are developed, it may be stated that there are the symptoms of pericarditis without the signs, the direct physical diagnosis of endocarditis being the recent development of valvular murmur. The patient often complains of a load about his heart, with dull pain, and frequently a sensation of heat. There is sometimes, too, a feeling as if the heart was too large, and its pulsations are generally, whether regular or irregular, of a greater force than could have been anticipated from the character of the pulse. In some cases we may observe a ringing metallic sound attending the contraction, at least in the early stages; but this sign must not be relied on unless in connexion with other symptoms. Dr. Hope observes, that when the circulation continues free, the action of the heart, stimulated by the inflammatory irritation, becomes violent and abrupt, and he holds that the increased extent over which it is perceptible is proportionate to this violence, rather than to the inflammatory turgescence of the organ, as Bouillaud had supposed. In this opinion I entirely agree. In the more advanced stages we may have those signs of greater cardiac suffering which occur towards the close of fatal pericarditis, and I do not know any character by which they differ from that class of phenomena. It is not improbable that in some cases rupture of the chordæ tendineæ, as observed by Dr. Law, takes place in the advanced stages. Two causes concur to produce this terrible accident: one, the violence of the heart's action; the other, the brittleness of the tendinous chords themselves. Such an occurrence may be looked for, particularly when endocarditis attacks a previously hypertrophied heart.

We have seen that the occurrence of a valvular murmur is the most important physical indication of endocarditis, but we must inquire whether it be so constant a sign as that its absence would imply the non-existence of any such disease. In certain forms of pericarditis, where a serous or purulent secretion fills the sac, the attrition murmur may not be produced; and so in endocarditis it may happen that, whether owing to the nature of the inflammatory product, or to the fact that the valves escape alteration, there may be, for a time at least, absence of valvular murmur. The following case is worthy of careful study with reference to this question:—

CASE X.—*Symptoms of Carditis, Valvular Murmur being only occasionally developed; absence of Friction Signs; Death.*

A woman, aged 30, having been six days ill, was admitted into the Meath Hospital, with symptoms of fever, to which were added palpitation, pain, and oppression in the region of the heart. She was cupped on admission over the præcordial region, with considerable relief. On the next day her tongue was clean, and she had little or no fever, but complained of pains in the bones. On applying the hand over the region of the heart, a peculiar vibrating feeling was communicated. The beatings of the heart were occasionally regular, but with now and then a long intermission, while at other times they became irregular and rapid. During this latter state the sounds were short, equal, and sharp, and closely resembled those produced by a dog when rapidly lapping water. In this condition there was no bellows murmur, but when the slower and regular contractions supervened, a murmur, evidently endocardial, was developed. There was no friction sound, nor any dulness of the region of the heart. The patient was treated by local bleeding, blistering, and the use of mercury, and for two days improved, when she was suddenly attacked with general coldness of the surface, rigidity, and slight delirium; the pulse was feeble and indistinct, with occasional long intermissions, its rate about 130. The sounds had the same lapping character as before, with a distinct thrilling impulse; there was still no friction, nor any endocardial murmur.

Notwithstanding the use of antispasmodic and gently stimulating medicines, and the mercurialization of the system, which was effected by inunction, the symptoms continued, attended by two new phenomena: one, the doubling of the second sound, and the other, a continued sensation of sinking or faintness about the heart. The double second sound was very feeble; the stomach became extremely irritable, and she complained of huskiness and loss of voice. The throat was neither sore on pressure nor swollen; the countenance became sharp and sunken, with a flush in the cheeks, and she died on the twenty-first day of her illness, the phenomena referrible to the heart having continued up to her death, without any change from the ninth day. No dissection was obtained.

No one can doubt this was an instance of carditis; yet there was no murmur produced, except at the earlier periods of the case, and that, too, in a transitory manner, only perceived when the heart was in an interval of comparative repose. Careful examination during life showed that there was no pericarditis, so that the case may be taken as an example of endocarditis, in which murmur disappeared long before death.

Let us recapitulate the facts of this case.

First, Alternations of slow and nearly regular action, attended by murmur; with paroxysms of rapid irregular action, but without any endocardial murmur.

Second, The latter character becoming constant.

Third, The feebleness of pulse, and doubling of the second sound.

It is probable that the occurrence of endocarditis without murmur, at least in its earlier stages, is of greater frequency than we have hitherto believed; and this may account for the appearance and advance of a valvular murmur after the cure of pericarditis. Such a case is not uncommon, and we may believe that, although during the early periods of the disease there existed no murmur, yet that endocarditis was silently forming, only to develope its signs when a certain amount of disorganization had occurred. Should this view not be adopted, we would be forced to admit, what seems improbable, that an endocarditis was developed after the subsidence of the pericarditis, and this in a latent manner, when the inflammatory condition had, to all appearance, passed away.

It appears probable, that when from any cause the heart becomes weakened, such as occurs under the influence of the typhoid state, or when a copious effusion exists in the pericardium, endocarditis may be present without murmur. This was, perhaps, the case in that example of inflammation of the pulmonary valves described by Dr. Graves, where the deposition of lymph on the valves, which were but two in number, was so abundant. The heart felt very soft, and lay collapsed; its structure was pale, and the pericardium was distended with straw-coloured fluid. There was extensive hepatization of the right lung.[1]

Finally, we might expect that the ordinary signs of endocarditis would be wanting in some cases of phlebitic disease; and it is possible that the small coagula, instead of being accumulated at the orifices, are, as Bouillaud and others have described, entangled among the fleshy columns. Here, however, although there is a mechanical change within the heart, it may not be competent to alter the currents of the blood in such a manner as to cause murmur.

The next case illustrates the effect of acute endocarditis in developing the signs of an old disease of the valves.

CASE XI.—*Dilatation and Hypertrophy of the heart; Ossification of the Mitral Valves, unattended by murmur; Supervention of Acute Endocarditis, developing a loud Murmur with the First Sound.*

A young man, who presented all the symptoms and signs of chronic emphysema of the lung, entered the hospital, labouring under great aggravation of his symptoms, induced by a recent attack of bronchitis. So great was the inflammation of the lung that the diaphragm showed signs of extensive depression, the pulmonary sound extending for nearly two inches below the ensiform cartilage. The heart was, of course, dislocated downwards, but no valvular murmur was discovered. After some time, the bronchial effusion becoming very profuse, but with great decrease in the volume of the lung, the patient was ordered small doses of turpentine, with tincture of lytta, which for a time produced some benefit. After a few days, symptoms of fever were developed, and the heart's action became greatly and permanently excited. A loud and hoarse murmur was now found to attend the first sound; it was most distinct over the region of the mitral valve, and was not propagated into the arteries; there was no friction sound, nor increase of dulness, and in this condition the patient continued up to the time of his death, which took place in a few days after the appearance of cardiac murmur.

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[1] Clinical Medicine, First Edition, p. 904.

The symptoms and physical signs, taken in connexion with the absence of all friction phenomena, led us to the diagnosis of acute endocarditis. On dissection, the heart was found generally dilated and hypertrophied; the aortic and pulmonary valves were of a deep red colour, and appeared softened and villous; the left auriculo-ventricular orifice was contracted by an extensive earthy deposit, causing great irregularity on its ventricular aspect, but forming a more regular deposit on the auricular side, the lining endocardium was generally red, but no lymph was detected on its surface.

The existence of considerable ossific deposits in the valves, yet without the production of murmur, is a fact well known to clinical observers. It is also ascertained that, for the production of murmur, we must have not only valvular alteration, but a certain degree of force in the action of the heart, so that we are sometimes obliged to excite the organ, in order to develope the signs of a disease which otherwise might be wanting. The excitement of the heart, however, by an endocarditis, has not hitherto been enumerated as among the causes for the production of a murmur under these circumstances.

From what has been now said, we may draw the following practical conclusions:

1. That endocarditis is a disease more frequently met with in combination with pericarditis than as an isolated affection.

2. That it may arise simultaneously with pericarditis, constituting the true endo-pericarditis; it may follow, or sometimes precede, the inflammation of the pericardium.

3. That the tendency to its production in rheumatic fever must be considered less decided than that of pericarditis.

4. That its symptoms can scarcely be said to differ from those of pericarditis.

5. That there is no pathognomonic sign of its existence.

6. That its diagnosis depends on the recent production of a valvular murmur under circumstances indicative of cardiac irritation, or the existence of special morbid states of the system, which predisposes to inflammation of the heart.

7. That where the symptoms of pericarditis are developed, but with absence of attrition sounds, or evidences of pericardial effusion, we may make the diagnosis of endocarditis, especially if there be the recent development of valvular murmur.

8. That the development of valvular murmur is not necessarily a consequence of this disease, at least in its more acute stages.

9. That the causes which in some rare cases of pericarditis prevent the production of attrition sounds may be supposed also to act in endocarditis. If the products of the inflammation be of a homogeneous nature, if they be purulent or merely sanguineous, and if, moreover, they form no depositions on the valves, an endocarditis may exist without the production of any valvular murmur.

10. That the causes which tend to prevent valvular murmur, even in extreme and chronic diseases of the valves, may also act, in cases of acute endocarditis, in producing the same result. Of these the two most likely to occur are, the weakness of the heart itself, and the over-distention of its cavities with blood.

11. That endocarditis, in consequence of its effect in exciting the muscular contractions of the heart, may actually develope a murmur, part of which, at least, proceeds from former and latent chronic disease.

12. That, although many cases of valvular disease evidently spring from an endocarditis, yet that we are by no means justified in attributing all valvular lesions to this cause, nor are we right in considering and treating such cases, even when they become chronic, as examples of chronic inflammation. It is true that the first morbid changes may have been inflammatory; but this state ceases, and is succeeded by new pathological conditions of deposition and transformation of tissue.

13. That we cannot distinguish between endocarditis affecting the right side of the heart and that of the left cavities.

14. That in cases of endocarditis passing into chronic and progressive disease of the valves, we are not able by physical signs to indicate the period when the inflammatory process changes into one of mere transformation and deposition.

MYOCARDITIS.

Our knowledge of the effects of inflammation in altering the muscular structure of the heart is still extremely limited; and we can easily understand why the pathological anatomy of myocarditis should be so scantily illustrated, as compared with that of inflammation of the pericardium or lining membrane of the heart, when we reflect that paralysis of muscular fibre appears to precede its disorganization. If this paralysis affect any considerable portion of the heart, death occurs before there is time for structural change. It is only, then, in cases either of a local myocarditis, or in those where the inflammatory action has concentrated itself upon a point, that we can study with advantage the anatomical character of the disease. Of myocarditis, independent of inflammation of the pericardium or endocardium, it may be safely said that we know nothing; at the same time we would not be justified in denying the possibility of its existence.

Myocarditis may be studied, first, as occurring in cases of the preponderance of pericarditis, and next, in those where it appears to spring from inflammation of the endocardium. From the very limited acquaintance I possess of this condition, I would say that it is most likely to be manifested in those cases in which, upon an attack originally of the highest degree of acuity, a true chronic inflammation has succeeded. This is especially true in the pericarditic cases; and it appears probable that in such the external layer of muscles is the first to exhibit perceptible anatomical change. On the other hand, the examples of internal ulceration of the heart may be supposed to arise in connexion with intense endocardial inflammation.

Of the first of these I have seen a single instance: the patient, a youth aged about 18, after being excited and overheated by violent gymnastic exercise, slept for several hours, lying on his left side on the cold, damp grass; he awoke in a state of collapse, attended by pain in the præcordial region, so severe as to awake him. More than a week elapsed before I was called to see him. On my first visit he presented all the symptoms and signs of the most violent pericarditis, and this condition, though somewhat mitigated, remained until the patient's death. No treatment which was adopted seemed to have the slightest effect in controlling the disease. The patient suffered in an exaggerated form all the miseries of a violent cardiac inflammation, and had the most indescribable and persistent anguish. On dissection, the pericardium contained a quantity of coffee-coloured, sanious fluid, mixed with shreds of coagulable lymph. The serous membrane was covered thickly with a dark-coloured, false membrane, so disposed as to give a generally honeycombed appearance to the entire surface of the heart. In numberless points ulcerative absorption of the serous membrane had taken place, and corresponding to these were well-defined depressions in the muscular structure, of two or three lines in depth, and of the same, or even a greater extent in diameter, evidently resulting from loss of substance in the muscle itself. The whole heart had a livid, almost black hue, which, however, decreased in intensity as we approached the inner layers and columnæ carneæ. I have no record as to the state of the endocardium.

The condition of the pericardium in this case was precisely similar to that which we observe in protracted cases of empyema and pneumothorax, where perforations of the serous membrane, not, as in the first instance, occurring from within outwards, but from without inwards, are found to exist, constituting a new order of secondary fistulæ.

This case, clearly an example of general carditis, would have been placed by Testa under his general head of Gangrene and Rupture of the heart (*Cancrena e Rottura del Cuore.*) According to him, the heart, like all other organs subject to local inflammation, may be attacked with the most violent form of the disease, eventuating in ulceration and gangrene, examples of which he points out in the works of some of the older authors. This writer, however, dwells especially on the gangrenous or ulcerative disease of the heart, which proceeds from internal inflammation. Thus he describes a case in which a careful examination of the right cavities, the ascending cava, and the pulmonary artery, showed a flocculent surface, as if the membrane was putrified. This was tinted by a black sanies, similar to that seen on the surface of gangrenous sloughs. In another case he describes the ulcerative process developed in the left ventricle, the carditis in this instance being apparently induced by long-continued and violent exertions. He gives a third case, which was probably one of endo-myocarditis, associated with a dissecting aneurism of the aorta. Death took place by rupture into the pericardial sac. A Bolognese lady, aged 28, of an ardent temperament and strong passions, was condemned to an imprisonment of fifteen years. The severity of her incarceration and mental excitement induced an inflammatory fever. Some months afterwards she was attacked with an internal sensation of cardiac suffering, attended with lancinating pain, with which she was at times, as it were, transfixed. This principally affected her during the act of eating, so that she frequently had to remove the unmasticated food from her mouth. She suffered from most violent palpitations, so severe that she frequently thought her last moments had arrived. Notwithstanding these sufferings, the patient lived for more than a year. During the last three months she had cephalalgia and vertigo. The face was pallid and livid. She had also acute, though fugacious pains in the chest, shoulders, arms, and loins. These symptoms were mitigated on the occurrence of a periodical epistaxis, which took place every fifteen days. Bleeding from the foot and other evacuations had also an alleviating influence. Her death was sudden: while she was in the act of speaking to her companions, she fell to the ground, and ceased to exist.

On dissection, the lungs were found to be healthy. The pericardium contained not less than two pounds of blood; and the heart, which was of a natural size, was flabby, and covered by a thick layer of fat. The anterior auricle was distended with blood, and greatly attenuated, while the posterior auricle was small and contracted. The anterior ventricle presented thickened and firm walls; it was without blood, but covered with deep ulcerations. The semilunar valves were for the most part destroyed by a vast ulceration, extending nearly to the arch of the aorta, and beyond this point to the bifurcation of the vessel in the abdomen; the lining membrane was of the brightest red colour; a large rent of the artery had taken place at its orifice.

In this case we have an example of a chronic ulcerative endocarditis and an aortitis. The fatal effusion of blood took place by the extension of the ulcerative process which had been established at the orifice of the aorta.

Such cases, however, are in these climates of rare occurrence, and the example now given from Testa is chiefly valuable as illustrating the greater violence of local inflammation which is met with in the warmer European climates, where, doubtless, both essential and local diseases are often developed in their highest degree of intensity. It is one of these rare cases, the principal value of which, in a practical point of view is, that it exhibits the extreme degree of lesions which are generally met with in a mitigated form.

According to Hasse, the existence of a general carditis, where all these structures are engaged, must be considered as a rare occurrence, at least when we speak of the disease in its highest degree. But he considers that the coincidence of the three forms in a minor degree is much more common, and he believes that none of the forms can occur in its highest intensity, without implicating the other textures of the heart. We are as yet but little acquainted with the pathological appearances of inflammation of muscular tissue, but serous infiltration, purulent softening, and abscess, appear to be the leading marks of the different stages of myocarditis. It is further stated by Hasse that myocarditis generally attacks the left ventricle, and my experience of the acoustic phenomena of severe pericarditis appears to confirm this statement, as we commonly find a failure of the first or systolic sound in the advanced stages of the disease. It is probable that in such cases both ventricles are engaged, but especially the left.

Abscess of the walls of the heart may be occasionally met with; but we must not confound a true phlegmonous abscess with the purulent deposits to which, in common with other organs, the heart is liable in cases of phlebitic disease. Professor Smith has met with some instances of apparently true inflammatory abscess of the heart; and a case is given by Dr. Graves, in which, in addition to the usual symptoms of hypertrophy of the heart with valvular disease, the patient suffered from violent pain in the region of the heart, darting over the chest, and which, towards the close of the case, became excruciating. His death took place suddenly. The heart was found greatly enlarged, and the pericardial sac was obliterated by adhesions, which, except at the apex, were easily broken down. In the latter situation they were strong and firm, and in the attempt to break them a rent was made in the substance of the heart, through which more than two ounces of purulent matter escaped. This rent communicated with a cavity in the substance of the heart, capable of containing more than two ounces, and lined with a firm cyst. The semilunar valves were greatly ossified.[1]

This case may have been originally one of general carditis, ending in the quadruple lesion of valvular disease, hypertrophy, abscess, and obliteration of the pericardium. How far the existence of the abscess may account for the character of the pain is worthy of inquiry. We not unfrequently observe pain in the ordinary cases of enlarged heart and permanent patency of the aortic valves, but in this case the violent, persisting, and paroxysmal character of the pain seems to indicate that it proceeded from some special cause.

We may, in the present state of our knowledge, arrange the results of myocarditis as follows:

1. An injected state of the cellular structure, followed by serous or sero-sanguinolent infiltration, and diminished consistence of the muscular fibre. (Hasse.)
2. Lardaceous transformation of the effusion, giving a homogeneous appearance to the structures; the muscular fibres, however, retaining their texture and form. (Hasse; Gluge.)
3. Interstitial suppuration, analogous to that in the advanced stages of pneumonia.
4. Abscess in the muscular structure of the heart.
5. Superficial ulcerations, presenting a crebriform appearance. These may be seen on the outer surface of the heart, in connexion with severe pericarditis, as in the case which I have detailed,

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[1] Clinical Medicine, Lecture xxxviii.

or on the inner surface, when there is a complication with intense endocarditis.

We have no means of diagnosticating any of the forms of suppurative myocarditis.[1]

There are other forms of disease, however, which, if not in every case to be attributed to carditis, appear often related to it. Of these, rupture of the valves, the occurrence of adherent coagula, purulent cysts in the heart, and partial aneurism of the ventricles, may be considered as examples.

I have never met with an instance of rupture of the chordæ tendineæ which could be attributed to acute endocarditis, but there is nothing impossible in such an occurrence. Hasse states, that in a few instances he has found the semilunar valves of the aorta and the pulmonary artery inflamed, and torn into shreds and filaments, which, covered with little wedge-shaped pellets of coagulum and effused matter, floated in the arterial tube in the direction of the current of the blood.[2] The same author speaks of the rupture of one or more of the papillary tendons, and observes that this accident is more common at the mitral valve. We are not, however, to attribute all cases of rupture of these chords to an acute inflammation, as doubtless the lesion more often results from the brittleness of the chords, which may be one of those changes occurring as a sequence of inflammation, though not with an actually existing inflammatory state.

Carditic Polypi.—On the occurrence of this form of disease pathological investigation has as yet thrown but a doubtful light; and, though according to Rokitansky, Bouillaud, and others, there is reason to believe that large polypi may result from carditis, yet the cases in which coagulation of blood in the cavities of the heart originates in a different manner are far more numerous than those in which it can be attributed to the effect of inflammation of the endocardium. But while we believe that carditic polypi or coagulations are not so frequent as Rokitansky, and especially Bouillaud, have taught, pathological analogy forbids us to deny that these polypi may result from carditis.

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[1] A case of purulent softening of the heart, by Dr. Salter, is quoted by Hasse. See Dr. Swaine's translation, p. 120. Dr. Swaine, in a note refers to a case by Mr. Stanley, Medico-Chirurgical Transactions, 1816, and to another quoted by Dr. Bennett, British and Foreign Medical Review, No. xxxix., which is taken from the Bulletin de l'Académie Royale de Médecine, Avril, 1843.

[2] Op. Cit., p. 130.

It is now some years since Dr. Graves and I published a case of very extensive arteritis affecting the right common iliac artery, and the arteries of the corresponding extremity. The patient had been attacked, about two months before his admission, with alternating sensations of burning heat in the toes of the right foot, followed by pain, coldness, and complete loss of sensation in the foot. In this condition he remained until the day of his admission, on which day the pain suddenly extended to the calf of the leg, and became intolerable, attended with nearly complete loss of power of the entire extremity. During the night the pain extended to the thigh. Next day the temperature of the limb was found to be about 58°. From the middle of the thigh to the toes, all sensation was lost; and, excepting that he could rotate the thigh slightly, there was no other voluntary motion of the limb. The femoral artery was felt to be hardened, and apparently enlarged; it was painful on pressure, and without pulsation.

Gangrenous action soon took place, speedily followed by death. The right common iliac artery was livid, and distended by a clot, which stretched into the external and internal iliacs, and all their branches, downwards, as far as they could be traced. The lining membrane of the vessel was red and villous, and in some portions the clot was separated from the vessel by a layer of dark-coloured puriform matter.

This case admits of more than one interpretation, but is interesting as being an instance of coagula in connexion with an arteritis.[1]

APPENDIX TO THE PRECEDING CHAPTER.

There are two subjects which may be noticed here, viz., the occasional doubling of one of the sounds of the heart, and the existence of purulent cysts within the cavities of that organ.

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[1] Report of the Meath Hospital, Dublin Hospital Reports, vol. v.

DOUBLING OF ONE OF THE SOUNDS OF THE HEART.

Among the physical signs of derangement of the action of the heart, I know of none more obscure in its nature than the doubling of one of the sounds. It is as if the sound, in place of being single, was divided into two sounds, in some cases similar in tone and duration, in others differing in both these qualities. This sign seems to affect the left more frequently than the right side of the heart, and in the majority of cases occurs in connexion with the second rather than with the first sound. We are not, I believe, as yet in a position to explain the nature of this phenomenon; but it appears more frequently to be connected with functional than with organic or inflammatory diseases of the heart. Analogy, however, would lead us to expect that this condition, like many other symptoms of functional affections, might be met with in connexion with inflammation, just as pain, irregularity, and palpitation are common to both conditions; and I have occasionally observed the sign in question in connexion with symptoms of endocarditis. Of this the following is an example:—

CASE XII.—*Symptoms of Acute Endocarditis; Doubling of the Second sound.*

A woman, aged 28, was admitted into the Meath Hospital, in January, 1840. She had enjoyed good health until a few days before admission, when she was attacked with rigors, prostration of strength, loss of appetite, and extreme thirst. Pain and palpitation of the heart set in, and she referred all her sufferings to that organ. No morbid physical sign could be discovered. Three days afterwards the pulse was 130, weak and intermitting, while the action of the heart was violent. A slight bellows murmur accompanied the first sound; she complained of a feeling *as if her heart was tearing out.* Two days afterwards, the following changes were found to have occurred: the bellows murmur had disappeared, and the second sound had evidently become double, and was much louder than the first: the action of the heart continued violent. In this state she continued for ten days, the heart all the time acting with great violence, the pulse rapid, and exceedingly feeble. She soon afterwards died.

In this case the dissection was not satisfactory, as the body was removed to a public dissecting-room, where the arterial system was injected from the aorta for the purpose of demonstration. The heart was not enlarged, but the wax injection had filled the left ventricle, in all probability by lacerating the valves. The lining membrane of the heart was of a deep red, with a purplish hue. The stomach was vascular, and presented the hour-glass contraction.

That this case was an example of carditis no doubt can be entertained. The patient had been in the enjoyment of good health up to the period of the first rigor, and the absence of signs of valvular disease on her first examination showed that the heart had been previously healthy. The pain, the cardiac anguish, the rapid and irregular pulse, the violent and jerking action of the heart, if taken in connexion with the absence of pericarditic signs, and the peculiar valvular phenomena, all indicate that endocarditis of a severe kind existed. The cessation of the mitral murmur, followed by the doubling of the second sound, is worthy of especial notice.

This patient was treated by local bleeding, counter-irritation, and mercury. Ptyalism was produced, but without any beneficial effect on the symptoms.

CASE XIII.—*Rheumatic Endocarditis; Distinct doubling of the Second sound.*

A young man, aged 16, was attacked with acute arthritis, in the month of August, 1838: his health had been previously excellent. On the day of his first attack he suffered from violent palpitations. He was admitted on the eighth day of his illness, with the usual symptoms of acute rheumatism affecting many of the joints. The pulse was 90, full and thrilling, with this form of irregularity—that after twelve or fourteen strong and full beats, three or four small, quick, and feeble pulsations would succeed. The impulse of the heart was strong, and the first sound was accompanied by the slightest possible bellows murmur. He was treated by the application of leeches to the inflamed joints, and to the cardiac region, with great relief; but in a few days the symptoms returned, the heart's action intermitted after every third or fourth beat: the first sound presented a distinct bellows murmur, while the second was replaced by two short, sharp sounds. Palpitations and pain were absent. By the use of leeches, counter-irritation, and digitalis, the cardiac symptoms were removed, and the patient was discharged free from any morbid state, except that the first sound of the heart was attended by a very indistinct murmur. This patient was admitted seven months subsequently. The rheumatic disease had returned, and produced all the usual effects of chronic arthritis. The heart's action was irregular, with a feeble impulse, and remarkably weak first sound, which had a dull, muffled character, with an occasional faint bellows murmur. The character of the irregularity was such, that the heart would occasionally beat for upwards of a minute without any intermission; then a distinct intermission would occur, followed by several quick, short pulsations. Although occasionally suffering from palpitation, he did not complain of any uneasiness about the heart.

CASE XIV.—*Arthritis; Cardiac complication; Bellows murmur accompanying the first sound; doubling of the Second sound while the patient remained in the horizontal position.*

A woman ætat 30, was admitted to the Meath Hospital, Oct. 31, 1839, labouring under an acute arthritic affection. At the time of her admission she was much prostrated, and suffered severely from pain in several of the large joints. Her pulse was 140, weak and intermittent. A loud bellows murmur accompanied the first sound of the heart, the impulse of which was abrupt and jerking. She continued in this condition for several days, no change being observed in the physical signs, as above described, until the 6th of November, when, on examining the heart, its second sound was found to be distinctly doubled; but the murmur still remained confined to the first sound. No improvement was at this time observed in her symptoms. The arthritic affection continued severe, with profuse perspirations and great nervous depression. In a few days, however, a marked change for the better took place, and this not only in the symptoms, but also in the physical signs. The impulse of the heart returned to its natural standard. The murmur decreased both as to loudness and prolongation, and the doubling of the second sound could only be distinguished when she assumed the horizontal position. From this period she gradually improved, and on examination of her heart, a few days prior to her leaving hospital, we could only detect the murmur with the first sound when she was in the recumbent position; the second sound was perfectly normal.

This case exemplifies the double second sound existing in endocarditis, and also its cessation when the patient was erect.

The doubling of one of the sounds of the heart cannot be considered as any special sign of any of the forms of carditis, for we meet it in cases of a different kind. It may be observed in nervous and chlorotic patients; and I have lately found it in the case of a man very far advanced in life, who was labouring under the symptoms of peripneumonia notha. It is, then, clearly only an indication of a special form of disturbance of the action of the heart. What its origin may be is difficult to declare; but that it is to be attributed to valvular rather than to muscular action appears more than probable. The greater frequency of its occurrence with the second sound, and the fact recorded in the last case, of its disappearance in the erect position, seem to point to this conclusion. I do not know of any condition which would be adequate to explain the occurrence in question, except a want of synchronism in the action of the pulmonary and systemic portions of the heart.

PURULENT CYSTS OF THE HEART.

In giving the results of my observations on this affection, I am desirous that it should not be believed that I am satisfied as to its nature, especially as to its being one of the results of carditis. The truth is, that great obscurity still hangs over the history of this affection, and it is here introduced rather as a matter of convenience than with any desire to promulgate the doctrine that carditis may produce this peculiar lesion.

It is found that in certain cases which are examples of acute or chronic disease of organs and structures often remote from the heart, the cavities of this organ present cysts, as it were entangled in its fleshy columns, and exhibiting various degrees of adhesion to its walls. Their size is various, and they generally contain a purulent fluid, which in some cases appears to be undergoing a process of transformation in which atheromatous or calcareous matter appears. They are to be met with both in all the cavities of the heart, and may be found in hearts otherwise healthy, at least so far as the endocardium is concerned, or exist with various forms of chronic disease, and even with purulent deposits, in the substance of the heart itself. (See Cruveilhier.)

Of the nature of this affection we cannot yet speak with any decision. We may, however, say, that it is not a result of ordinary endocarditis, inasmuch as the necessary conditions of this affection are often absent; and that the disease appears to want the symptoms and signs of ordinary inflammation of the heart. We will not here describe the different opinions put forward on the subject, but simply indicate a few of the most important. Three distinct doctrines are entertained on the point:

1. That they result from coagula produced by inflammation, which themselves take on a suppurative action. They may thus be considered as remotely the effects of endocarditis.

2. That coagula being formed, from whatever cause, they become purulent, owing to the existence of a pyogenic diathesis.

3. That they may be the result of a true cardiac phlebitis.

Many circumstances lead to the opinion that it is to the two latter causes that we should refer this peculiar condition: at the same time it must be confessed that the entire subject of the conversion of fibrine into pus is involved in extreme obscurity.

Even if it could be admitted that simple coagulation of blood was a common effect of endocarditis, there would be a great probability against the coagulum becoming the nidus of a purulent deposit. If we refer to the case of aneurism, in which successive layers of fibrinous coagula are formed, how rarely does it happen that they exhibit any purulent change. May not some analogy be supposed to exist between the coagulum found in the heart, and continuing after its exciting cause has been removed, and that met with in a large aneurismal sac?

A greater degree of probability exists in favour of the second supposition:—that a coagulum having been formed, either anterior to or consequent upon a pyogenic state, it becomes, in virtue of its feeble organization, or the action of some elective affinity, the nidus of a purulent deposit. Something analogous to this is seen in cases of arteritis, as already described; and in other instances, where the coagulum has been found not only surrounded by a purulent layer, but actually containing pus in its very substance.

Without denying that purulent cysts of the heart may in some cases admit of this explanation, Professor Smith inclines to the belief that they may result from a cardiac phlebitis. It is certain that they have been often found in cases of phlebitic disease, and as in such cases organ after organ seems to assume this special form of disease, there is no reason why there should not be a cardiac as well as a renal, hepatic, pulmonary, or uterine phlebitis. It is true that in many cases of venous inflammation a great number of organs become affected, but this is by no means constant; and the frequent exemption of this or that structure or organ inclines us strongly to the belief that the existence of purulent matter in particular situations is owing less to any general purulent state of the blood than to the production of a specific irritation in the organs so affected. We are still, however, in great want of further researches on the subject, but Professor Smith's views are strengthened by the fact already noticed, that in some of these cases of purulent cysts in the cavities, deposits of pus are to be found in the substance of the heart itself.

The fact of these purulent collections being found encysted would seem to connect them with the process of chronic disease. Of this the following case is an illustration.

CASE XV.—*Purulent Cysts in both Ventricles*: *Protracted symptoms of Phlebitic Disease.*

An Italian, after having for a length of time abstained from intoxicating liquors, had indulged to great excess in their use, and was admitted into the Meath Hospital, labouring under a complication of alarming symptoms. He had a low irritative fever, attended by symptoms of delirium tremens, and a feeble pulse, generally ranging between 130 and 150, and it is remarkable that this quickness of pulse continued to the period of his death, which happened two months after his admission. On one occasion it fell to 120, but soon resumed its former rate. In addition to these symptoms the left thigh and leg were extensively swollen, presenting the general appearance of the second stage of phlegmasia dolens. The right lung exhibited the signs of pneumonia in its inferior portion, with bronchial respiration at the root of the lung, and friction sounds laterally and anteriorly. These signs, as well as the crepitating râle, remained singularly persistent, notwithstanding the employment of such general and local remedies as the state of the patient would justify. The fever passed into a species of hectic, and the patient died in a condition of extreme anæmia.

The abdominal cava was found to contain a long coagulum adherent to the vein; its surface was rough, and on its being detached we found the corresponding portion of the vein red and villous. In the femoral vein was a similar coagulum, and the artery, vein, and nerve were agglutinated. The saphena was obliterated, and felt like a hard cord, and this obliteration extended as far as the vein could be traced. In the right ventricle we found some dark-coloured coagula and creamy matter, but the endocardium showed no sign of inflammation. A number of small, white tumours, which proved to be cysts containing pus, were found between the columnæ carneæ. The left ventricle, also, exhibited these cysts, three of which were of great size, and adhered very slightly to the parietes of the heart. The inferior lobe of the right lung was solid, and, when cut, very nearly resembled red granite. There was no abscess, but purulent matter, exactly similar to that in the heart, could be squeezed from every part of the cut surface. In the upper lobe of this lung, as also in the left lung, numerous isolated deposits of the same nature existed, the intervening tissue being healthy. The liver, spleen, and kidney, the joints, and voluntary muscles, exhibited no purulent deposits.[1]

Mr. O'Ferrall has, on two occasions, exhibited specimens of this disease to the Pathological Society. In one of these cases the patient, an adult male, was admitted into St. Vincent's Hospital, labouring under an attack of pleuritis, but also presenting symptoms of hypertrophy of the heart, with hæmoptysis, anasarca, ascites, and albuminous urine. There was nothing in the phenomena of the heart beyond the ordinary signs of hypertrophy. He died five months after his admission. On dissection, the organ was found greatly enlarged: it contained numerous cysts, generally of the size of a bean, while some were as large as a walnut; they were attached to the internal surface of the ventricles as well as of the auricles; their contents were various, some being filled with purulent matter, others containing a substance closely resembling the fibrine of blood, while in a third class the contents seemed intermediate between fibrine and purulent matter. One of the cysts contained nearly two drachms of pus, and their internal surface had a villous appearance. A gangrenous cavity existed in the upper portion of one lung, while a large portion of the spleen showed a deposit of a yellowish-white substance, similar to the fibrine of blood.

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[1] See the Transactions of the Pathological Society of Dublin, December, 1842.

In another case, observed by Mr. O'Ferrall, the specimen was taken from the body of a boy aged 16, who had laboured under disease of the heart and kidneys. The urine was pale, albuminous, and of the specific gravity 1·010. The region of the heart was dull, and there existed strong impulse, and a bellows murmur. On dissection, the kidneys were found to exhibit Bright's disease in a certain degree. In the cavities of the heart several cysts, containing puriform matter, were found in the left auricle, and engaged among the fleshy columns of the right ventricle.

As to the nature and causes of these purulent cysts, it will be sufficient to say, that two opposite opinions have been defended by pathologists. One is that adopted by Mr. O'Ferrall, who holds them to be examples of purulent softening of clots previously formed; and the other that of Bouillaud, who considers the coagulation of the blood as the second step in the process. He believes that pus, carried into the cavities of the heart, there acts in producing coagulation of the blood. There is strong reason for adopting Mr. O'Ferrall's view at least in certain cases, for the instances he has brought forward of cysts containing a variety of contents, which were of the nature of decomposed blood in various stages, are most important. Still, however, the general history of these cysts is open to further investigation.

We are not yet in a position to declare the diagnosis of this lesion. In one of Mr. O'Ferrall's cases there appeared no physical sign of disease of the heart of any special kind; and in another, where organic disease affecting the valves existed, the signs presented no unusual character. It is greatly to be doubted whether we have any meanś of detecting an ordinary coagulum of blood in the heart, but we are not to despair of yet discovering some signs indicative of this accident.

The following case is worthy of being recorded. A young man, who had been previously in good health, was attacked with the symptoms of malignant cholera, during the last epidemic of that disease in Dublin. Within a very few hours after collapse had been established, a loud bellows murmur was discovered at the upper and middle sternal region. This continued up to the time of death; and on dissection a large coagulum was found in the left ventricle, stretching upwards, and extending through the aortic orifice into the arch of the aorta. The valves of the heart and its walls were found perfectly healthy, so that there can be no reasonable doubt that the bellows murmur was of recent production, and was owing to the interference of this remarkable coagulum with the proper action of the aortic valves.[1]

The occurrence of these purulent cysts in the cavities of the heart constitutes one of the most singular circumstances in the whole range of cardiac pathology. In the dearth of information on the subject, it will be desirable to state generally such observations as have been made upon it in Dublin. We find, that as yet no satisfactory explanation has been given as to the formation of these cysts, at least so far as the mechanism of the process is concerned. How a cyst, which in some cases appears to have no organic connexion with the endocardium, may be formed within the heart is still a matter of pure conjecture. We find such cysts entangled with the fleshy columns, yet without any connecting tissue or structure; while in other cases there appears to be an adhesion or slight organic connexion. Their contents are various. They may present decomposed blood in various stages, as Mr. O'Ferrall has shown. They may be filled with true pus, as in the case I have given, and also in the example recorded by Dr. Bigger,[1] in which the patient died of phthisis pulmonalis without ever having presented any symptom of cardiac disease. The cysts in this case were numerous, each about the size of a small bean, some of them merely inserted between the carneæ columnæ, others imbedded in the muscular substance. Lastly, as in a remarkable specimen preserved in the Museum of the Richmond Hospital, they may exhibit the cretaceous transformation of their contents. The cysts exhibit little, if any traces of organization, and so far as we know the disease, appear to affect both sides of the heart indifferently.

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[1] Owing to the kindness of Mr. Rynd, under whose care this patient had been, I was enabled to exhibit the post-mortem appearances to the Pathological Society. The case is one full of interest.

It is remarkable that while Hasse[2] declares that the purulent coagula of the heart occur oftenest at the left side, yet that Forget comes to the opposite conclusion;[3] and so far as the nature of the disease is concerned, we can form no other opinion, but that it is in some way connected with the pyogenic state. That it cannot be considered as one of the results of simple endocarditis is certain; and we know of no means by which its existence can be determined. A remarkable case is given by Forget, in which the cysts were confined to the left ventricle. The lungs contained many tuberculous ulcerations. In the case which I have recorded, and in the examples given by Mr. O'Ferrall, the cysts existed in both ventricles, a circumstance which goes to strengthen the opinion of Forget, that in the case which he has recorded, the limitation of the disease to the left ventricle was owing to the fact that the lung had supplied the purulent matter.

Before we conclude these general observations on carditic disease, we must allude to two points of importance in practical medicine: one of these is the innocuousness, even for many years, of valvular disease sufficient to afford prominent and permanent physical signs, and the other, the development of the signs of progressive chronic disease in a manner almost sudden.

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[1] See the Transactions of the Pathological Society of Dublin, 1838, Dublin Journal of Medical Science, First Series, vol. xv.

[2] Anatomical Description of the Diseases of Circulation and Respiration, by C. E. Hasse, Dr. Swaine's translation, London, 1846, p. 156.

[3] Précis Théorique et Pratique des Maladies du Cœur, Strasbourg, 1849.

It appears certain, that in some cases, after a valvular lesion has been established, the processes of organic change are either wholly arrested, or advance with extreme slowness, so that, should the condition of the heart's cavities remain unaltered, and the general health of the patient continue good, no symptoms of heart disease will occur for many years, and the individual may not only enjoy an apparently perfect state of health, but be able to undergo violent and fatiguing exercises, and even indulge freely in the use of stimulants. Such cases may go on for many years without the occurrence of any symptom which awakens the attention of the patient, or excites the apprehensions of the physician. Yet all this time a valvular murmur has existed in the heart.

Now, it may often happen in such cases, that the patient, having contracted some inflammatory affection of the lungs, consults a physician who has had no knowledge of his previous history. A stethoscopic examination is made, a loud murmur is detected, and a twofold error is commonly committed: first, that the murmur is supposed to indicate a recent and progressive disease, and next, that the patient is suddenly, and for the first time informed, that he has an organic disease of his heart. Physicians who cannot help thinking aloud, or who, less excusably, are fond of exhibiting their diagnostic tact to the patient, are but too apt to commit these errors. The greatest evils now result, for the chief safeguard of the patient is at once removed, and his attention is painfully directed to the state of his heart, than which there could be nothing better calculated to hasten its disease. But this is not all: a long-existing change, which we might compare to the cicatrix of a wound, is taken for a recent and progressive disease. All the habits of the patient are altered by peremptory mandates; he is debarred the use of wine; he is placed on a low diet, and all action, exercise, and pleasurable excitement are forbidden. The discoverer of the disease, too, must now attempt to cure it. Local and general depletion, mercury, digitalis, prussic acid, blisters and issues, are summoned to lend their aid in attempting an impossibility, and in doing that which ought not to be done, namely, weakening the heart, and exhausting the general nervous energy. Under such circumstances, and with the fear of sudden death continually before the mind, the results are just what might be expected: the action of the heart becomes enfeebled and irregular: its cavities dilate with or without hypertrophy; and dropsy and visceral congestion close the scene. I know of no case more aptly illustrative of the evils of the *nimia diligentia medici*.

The practical rule obviously should be, that when we accidentally discover a valvular murmur in the heart of a patient, whose previous health had been good, and who did not present any of the symptoms of disease of the heart, we should be slow indeed in communicating the fact to any one, least of all to the patient himself. We must, without exciting his apprehensions, seek to discover whether this murmur be the result of some long-previous illness, or whether it be of recent origin: and if it appears that the patient, during the past seven or ten years, had suffered from rheumatic fever, with or without the symptoms of carditis, we may with great probability conclude, that the disease originated on the occurrence of that affection. We must then examine into the habits of the individual during the period in question, and be very slow in advising any alteration in them, for common sense must teach us, that any system of living which had preserved the muscular portions of the heart from lesion, while the functions of the organ remained in a state of health, and which had not caused any advance in the valvular affection, should not be lightly departed from. And, above all, we must avoid the unpardonable error of treating a fixed and incurable organic change as a recent and progressive disorganization.

With reference to the second of the points above indicated, namely, the unexpected appearance of physical signs of chronic disease within a short space of time, we shall here content ourselves with the statement of the fact, reserving its full consideration until after the diagnosis of valvular disease is examined.

CHAPTER II.

DISEASES OF THE VALVES OF THE HEART.

It would be foreign to the purpose of the present work to enter into the long-agitated question of the causes of the heart's sounds, or to review the many conflicting opinions which have been put forward on this subject. I have been long convinced that in each series of observations on this point there was a source of error, namely, that an attempt was made to explain the sounds and impulse of the heart by reference to too limited a number of possible causes for their production. Thus, some have taught that the sounds depended upon valvular tension; some, on muscular contraction, and others, on the impulse produced by the current of blood. But if we reflect on the number of physical circumstances which, if not all concurring to produce the double stroke of the heart, must take place in the short interval of time occupied by each complete action of the organ, indicated by the arterial wave, we shall find that the number of operations or possible causes of sound is very great. We have—

1. The auricular contractions.
2. The ventricular dilatations.
3. The ventricular contractions.
4. The auricular dilatations.
5. The opening of the auriculo-ventricular valves.
6. The opening of the arterial valves.
7. The closure of the auriculo-ventricular valves.
8. The closure of the arterial valves.
9. The entrance of blood into two auricles.
10. The entrance of blood into two ventricles.
11. The exit *per saltum* of the blood from two ventricles.

So that we have here not less than twenty-two operations, which, however, if the heart is acting with regularity, may be reduced to eleven, in consequence of the simultaneous action of the pulmonary and systemic portions of the heart.

It is certainly not proved that every one of these operations produces sound. For example, we have no evidence that the relaxation of a hollow muscle is attended with sound. Still, even at the moment of this relaxation, a possible cause of sound exists in the impulse of the blood against the walls of the cavity: as occurs in aneurism from the entrance of the wave of blood into the sac.

It may, however, be assumed that in the regularly acting heart some of these operations have so much more to do with the production of the sounds than others, that they should be considered the principal, if not the only sources of the double sound: so that, for practical purposes, we may admit that the first sound corresponds to the ventricular systole, the second to its diastole. But coincident with both these conditions there is a valvular tension: in the ventricular systole the mitral and tricuspid valves are forcibly closed, while in the diastole the same condition is produced in the semilunar and pulmonary valves. It is not yet determined how much of the first sound depends upon muscular contraction, or how much on valvular tension; but this at least is certain, that, where the muscular contractility of the heart is impaired, it is the first sound that suffers most diminution. It is probable that in the production of both sounds there is the double source of muscular contraction and valvular tension; but that the former has a greater share than the latter in the production of the first sound; while, conversely, valvular tension has a greater share than muscular contraction in the production of the second. The first of these suppositions is, at all events, strongly confirmed by the fact of the failure, or even complete cessation of the first sound, in certain cases of typhus fever, attended with softening or weakening of the ventricles.

Indeed the fact of the heart's action continuing without a first sound might lead to the opinion that valvular tension had no part in the production of the sound. But it must be recollected that in such cases the closing of the auriculo-ventricular valves cannot take place with the same force as when the heart has full contractile power, so that the valves are, as it were, shut silently.

We have thus, as the principal causes of the acoustic phenomena of the heart's action, three conditions, namely, the contraction of its muscles, the closing of its valves, and the current or wave of blood passing from one cavity into another. These are, at all events, the sources of what may be termed the intrinsic phenomena of the heart's action, and have special reference to the production of the first sound. The second sound, or that produced by the arterial valves, on the other hand, may be termed extrinsic, and has relation to the motion of the blood after its departure from the heart.

But it is obvious that the first three, or intrinsic phenomena of the heart's action, will be strong or weak, manifest or obscure, in proportion to the strength or vivacity of the contractile force of the heart, so that the character of these intrinsic cardiac actions must depend on the vital force of the organ. *Cæteris paribus*, the sound produced by the contractions of the cavities of the heart, as well as that caused by the closing of the mitral and tricuspid valves, and the sound, if any such there be, produced by the current of blood, will be strong or feeble in proportion to the vigour of the heart.

With these views, we should expect to find the second sound or the extrinsic phenomenon less influenced by the condition of the heart than the first. We of course exclude from this consideration cases of organic disease of the semilunar valves. Experience shows that alterations of the second sound are rare, compared with those of the first, a circumstance which we should expect, when we call to mind the low degree of organization and simple structure of the arteries, as contrasted with that of the muscular apparatus of the heart.

Compared with the arteries, the heart may be held to stand in the relation, physiological, and anatomical, of a red to a white-blooded animal: and, pathologically, it is liable to a vast number of functional diseases; to every form and result of inflammation, except, perhaps, gangrene; to hypertrophy, atrophy, and numberless organic changes. The arteries, on the other hand, fulfil a less active function; their sympathies are but slightly marked, and their diseases are more frequently those of deposition and transformation than of active inflammation. But they appear to be the governors of the extrinsic phenomena; and hence these, or their representative, the second sound, are rarely altered, as compared with the first class of signs, which embraces the impulse and the first sound.

It will be seen by referring to the chapter on the condition of the heart in Typhus Fever, that in by far the greater number of cases of alteration or suspension of one of the sounds, that sound was the *first*, and that in many instances so complete was its obliteration, that the double action of the heart appeared suspended, nothing remaining but the second sound. I have suggested, that in the rare cases in which the latter becomes feeble, there is a diminution of the arterial force; but future observations must determine whether this be owing to any alteration of the vital contractility of the vessels, or of their elasticity alone.

It is, then, in the vital and anatomical conditions of the muscular fibre that we find the key of cardiac pathology; for, no matter what the affection may be, its symptoms mainly depend on the strength or weakness, the irritability or paralysis, the anatomical health or disease of the cardiac muscles. It was long ago observed by Laennec that valvular diseases had but little influence on health when the muscular condition of the heart remained sound, and every day's experience confirms this observation. We may extend it to many other cardiac affections, at least so far as the production of characteristic symptoms is concerned. Pericarditis without irritability of the muscle is often so completely latent as only to be discoverable by physical signs; and the same may, doubtless, be said of endocarditis; while it must never be forgotten that the important symptoms of these affections, as laid down in books, have reference to lesions of either muscular action or structure.

The difficulties which the diagnosis of valvular disease presents to the student, have been greatly increased by the conflict of opinion as to the nature and causes of the sounds of the heart, and by the various and opposite diagnostic rules laid down by writers, according as they incline to this or that theory. Let us endeavour to strip the subject of some of these difficulties, and to present it as a guide sufficiently trustworthy for all practical purposes.

It too often happens, when the existence of a valvular disease is determined, that great labour is expended in ascertaining the exact seat and nature of the affection. Long and careful examinations are made, to determine whether the disease exists at the right or left side of the heart; whether it be a lesion of the mitral, tricuspid, or the semilunar valves; a contraction or dilatation; an ossification; a permanent patency, or warty excrescence. Now, though in some, we might say in many cases, these questions may be resolved with considerable accuracy, it is also true that in a large number their determination is of comparatively trifling importance: and the two great practical points to be attended to are, first, whether the murmurs really proceed from an organic cause, and next, what is the vital and physical condition of the muscular portions of the heart; for it is upon these points that prognosis and treatment must entirely depend. There is, indeed, no other organ whose affections more fully illustrate the truth of this principle, that in dealing with the diseases of adjacent structures, diagnosis is easy where it is important, and of little value where it is difficult or impossible.

Another source of the difficulties with which this subject is surrounded is, that rules of diagnosis are in many cases founded on the supposition of the isolation of disease; but every practical man knows that in chronic diseases of the heart isolation is the exception, and complication the rule. Hence, one reason why disease at the bed-side so rarely corresponds with its description in books. Its combinations vary infinitely in their nature and number; and we often find, particularly in cardiac disease, that it is the more recent and least developed affection that produces the most prominent physical signs. Hence, in many cases, while we recognise a particular disease, we are unable to say whether another and even more important affection co-exists.

We should by no means underrate the importance of differential diagnosis in valvular disease: but the number of cases in which it is desirable to determine the exact seat and nature of the affection is comparatively small. Let us take the two most ordinary forms of this disease, namely, the insufficiency, with contraction on the one hand, and dilatation on the other, of the mitral and aortic valves. Certain rules of treatment are supposed applicable to each of these affections; but the truth is, that no constant state of the heart's muscles is attendant on them respectively, and it is mainly on the vital and mechanical conditions of the cavities of the heart that we can found any rule of treatment.

Perhaps more value attaches to the question when considered in relation to prognosis. In mitral-valve disease there is a greater probability of sudden death than in the analogous affection of the aorta; but if the cavities be yet unaltered, and the heart's action tranquil, there is in this disease a better chance of prolongation of life than in that of the semilunar valves, for this latter affection commonly leads to hypertrophy and dilatation of the left ventricle. It will not be out of place to remark, that sudden death in disease of the heart is by no means so frequent as is generally supposed. In the great majority of cases, death occurs in no sudden or extraordinary manner. It is principally in examples of solutions of continuity, such as the rupture of an aneurism, the laceration of the ventricles, or the breaking of the chordæ tendineæ, that this happens. We may add to this list a few cases of the fatty degeneration of the heart in which, without rupture, death takes place by a sudden syncope or a congestive apoplexy. But these are the exceptions, and in the greater proportion sufficient notice is given of the approach of death by long-continued symptoms of dropsy, and of pulmonary and hepatic disease.

So general is the belief that sudden death is the inevitable termination of disease of the heart, that the very suspicion of the existence of such an affection leads to great and injurious mental depression on the part of the patient, and corresponding anxiety among his friends. It will, therefore, be right that the physician, by appealing to the real facts of the case, should do his best to diminish those apprehensions.

Cases of valvular disease are of two kinds, those in which a carditis has been manifestly the source of the affection, and those in which we cannot trace the disease to any distinct attack of inflammation. In many of the latter the nature of the disease, as Hasse and others have taught, is analogous to the atheromatous and ossific affections of the arteries. And even in the first class, after disorganization of the valve has taken place, and the disease has become chronic, we have no reason for believing in the existence of even a chronic inflammation, and it is certain that we gain nothing by treating such diseases as examples of chronic carditis.

The various effects of organic disease on the function, structure, and form of the valves, is described in every work on pathological anatomy. In a practical point of view, it would be sufficient to recognise contraction or dilatation of the orifices, both of which conditions are attended by a permanently open state. This permanent patency is in some cases produced at an early period of the disease, while in others the valves may be so roughened by cartilaginous and ossific growths as to cause a murmur during the exit of the blood, while they yet remain competent to close the orifice. To this consideration we shall return, as it is one of those which may be indicated as opposed to over-refinement in diagnosis.

Valvular murmur is so much more frequently developed at the left than at the right side of the heart, that it is still a question whether we are in any case in a position to declare the existence of disease of the tricuspid or the pulmonary valves. If the relative position of the heart were always the same; if we had to deal only with cases of valvular disease, uncomplicated with change in the figure or volume of the heart; and lastly, if the rule were certain that the loudest sounds were to be found at the exact situation of the disease which produced them, it would be nearly as easy a matter to diagnosticate valvular disease at the right as the left side of the heart. But when we know that the investigator can seldom meet a case so circumstanced, and then reflect on the greater frequency of diseases of the left side, it becomes plain that the cautious physician ought not to commit himself hastily in a diagnosis of disease of the valves on the right side, much less declare its exact nature.

For it appears certain that we must be guided in our treatment of valvular disease less by the condition of the valves, than by that of the muscular portions of the heart. The practical physician, having satisfied himself that a valvular disease exists, will not devote too much time in ascertaining its exact nature; but he will examine into the vital and mechanical state of the heart's cavities. He will ascertain the amount of vigour of the heart; whether its force is above or below the natural standard; whether it is liable to excitement from slight causes; and whether irregularity of action or the opposite is its ordinary state. He will endeavour to determine the duration of the disease and its origin, and examine how far the brain, lungs, or liver, have suffered from the mechanical or vital effects of disease of the heart. Thus he will obtain some rule of treatment, and as the two most common diseases of the orifices, viz., permanent patency of the aortic and mitral valves, when occurring in an isolated form, are not difficult to distinguish, he will, so far as treatment and prognosis are concerned, be able to give to the patient all the advantages which the present state of medicine can afford.

In order to present this matter plainly before the reader, stripping the question of whatever is doubtful or unascertained, we shall suppose a certain number of cases or examples in which such a diagnosis as appears justifiable and of practical utility may be made.

UNCOMPLICATED DISEASE OF THE MITRAL VALVES.

Permanent murmur, with the first sound loudest towards the apex and to the left side, and not heard in the artery; second sound natural.—In this combination we have the common indications of organic disease of the mitral valves. The character of the murmur varies in different cases, and the sign may be distinguished, in most instances at least, from that produced by disease of the semilunar valves, in its being louder towards the apex than the base of the heart. It may be a smooth bellows sound, or present a grating character, with or without musical tone, and fremitus may or may not be present.

Now if the heart's action be regular, if the pulse have its natural fulness and character, if the impulse of the heart be not excited, we may consider such a case as an example of uncomplicated mitral-valve disease. If, on the other hand, the action of the heart be tumultuous and irregular, if the pulse be feeble and unequal, and the lungs show symptoms of congestion, we may suspect that the orifice is contracted and the heart otherwise diseased.

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DISEASE OF THE AORTIC VALVES, WITH PERMANENT PATENCY.

The first sound unattended with murmur; the second replaced by a murmur which can be perceived to be double; this murmur is more or less audible along the course of the aorta, and, as regards the heart, is generally louder at the base than towards the apex.— The phenomena now described belong to disease of the aortic opening, and indicate that regurgitation into the ventricle takes place, owing to the defective condition of the valves. Such cases may be divided into two classes; those in which the disease is in an early stage, and those much more chronic, when the usual consequences of a hypertrophied and dilated ventricle have supervened. In the first case there may be no evidence of enlargement of the heart, and the characteristic visible bounding pulsations of the arteries may not be developed, or only seen in the neck. But in the more advanced periods we have, in addition to the loud double murmur at the aortic orifice propagated into the aorta and large arteries, the remarkable symptom of the visible pulsations of not only the great trunks, but of many of the smaller arteries which approach the surface. The radial pulse becomes quite characteristic. This is the jerking pulse, "the pulse of unfilled arteries" of Dr. Hope,[1] and we have no difficulty in recognising an enlargement of the left ventricle, if not of the entire heart. We owe the diagnosis of this disease to Dr. Corrigan.

DISEASE OF THE AORTIC VALVES, WITHOUT PERMANENT PATENCY.

The action of the heart slow, feeble but generally regular, or only occasionally intermitting; a murmur with the first sound; the second sound healthy, yet a single murmur existing in the aorta and its large

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[1] Dr. Hope observes, that this character of pulse is produced by aortic regurgitation, in other cases as well as those where the reflux is into the left ventricle. He instances cases of communication with the pulmonary artery, or the mouth of the left ventricle. I have noticed this symptom in a case of true aneurism of the ascending aorta, in which the valves were competent to close the orifice. The name of collapsing pulse would be more appropriate, as the sensation given to the finger is that of a sudden disappearance of the arterial wave, which, as Dr. Corrigan has shown, is produced by the retrograde motion of a portion of the blood. See his original Memoir, Edinburgh Medical and Surgical Journal, April, 1832.

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branches.—This case, which is not unfrequent, would seem to justify the following diagnosis: Disease of the aortic opening causing murmur during the exit of the blood; the valves, however, being able so to close as to prevent regurgitation. To this may be safely added, that the heart is weak, and that in all probability this weakness proceeds from fatty degeneration. Indeed, when the pulse falls below 50 we may make the double diagnosis with considerable certainty.

Here the aortic valves are diseased, but not permanently patent. Hence, there is no regurgitant murmur, and we have the second sound unaffected, because the valves close more or less perfectly. The aortic murmur is propagated from the origin of the vessel, where it arises during the exit of the blood. This curious combination I have already described in a memoir on slow pulse,[1] and it is more than probable, though I cannot confirm this by recorded observations, that the murmur in such cases will be louder at the base than at the middle or the apex of the heart. We have thus produced from organic causes that group of acoustic signs which is often observed in anæmia, namely, the triple combination of *a murmur with the first sound, a clear second sound, and yet a murmur in the aorta.* When, however, all the circumstances of the case are considered, and especially when the coexisting signs and symptoms of a degenerated left ventricle are taken into consideration, there will be but little difficulty in coming to a correct conclusion as to the nature of the disease.

Such are the cases in which special diagnosis of valvular disease may be safely made. It is laid down by Dr. Hope, that the regurgitant diseases of the pulmonary and tricuspid valves may be made by applying the necessary inversions. Thus, according to him, the signs of diseases of the tricuspid valves are the same as those of the mitral, except that the murmurs are loudest on or near the sternum, at the same level as in the case of the mitral disease, namely, about or a little above the apex of the heart; and except, also, that the pulse is little affected with irregularity. But anatomical considerations should make us cautious in admitting these statements.

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[1] Dublin Quarterly Journal of Medical Science, vol. ii. 1846.

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Again, he observes, that when there is regurgitation through the valves of the pulmonary artery, a murmur accompanies the second sound; its nature and diagnosis are the same (the necessary inversions being made) as in the case of aortic regurgitation, except that the pulse is not jerking. A purring tremor has been found to attend dilatation of the pulmonary artery.[1]

It has been already observed that the practitioner should use great caution in giving a diagnosis, not only of the nature, but of the very existence of valvular disease at the right side of the heart; and Dr. Hope himself has dwelt on the necessity for the exercise of this caution, and pointed out that the signs he has specified must be perfectly well marked to justify the opinion. But although, in the last edition of his work, this excellent observer has not dwelt so strongly on the attainable certainty of special diagnosis in valvular disease, he still, I think, underrates the sources of difficulty that must accompany all attempts to discriminate the valvular diseases of the right side of the heart.

DILATATION AND FEEBLENESS OF THE HEART, WITH OR WITHOUT VALVULAR DISEASE.

The heart's action permanently irregular, with an extended, but not a strong impulse; the sounds so rapid and unequal that their analysis is difficult, rendering it often impossible to distinguish the first from the second sound; murmur generally absent; the pulse rapid, feeble, unequal, irregular; no aortic murmur; signs of pulmonary and hepatic congestion.—This is one of the cases of heart affection to which the practitioner's attention will be most commonly directed; and though valvular disease is by no means a necessary attendant upon it, it is introduced here because it is considered to be almost always accompanied by some form of that affection. Valvular murmur is generally absent, or it may exist for a time and then disappear; and it is certain that no constant morbid state of the valves attends the disease. The orifices may be dilated or contracted. It occurs in gouty and debilitated habits, and is almost always attended with chronic bronchitis and enlargement of the liver.

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[1] See Hope.

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The diagnosis in this case is to be, that the heart is generally thinned, dilated, and weakened, the probabilities being strongly against the existence of any important disease of the valves. To the consideration of this disease we shall return.

EXTREME OSSIFIC DISEASE OF THE AORTIC ORIFICE.

Strong action of the left ventricle; extremely loud and musical murmur at the aortic orifice, transmitted through the whole extent of the arterial tree; the heart's action generally regular.—I have witnessed two or three cases of this combination. The phenomena arise from extensive ossific disease of the aortic opening, which is rendered not only rigid, but singularly irregular, from the deposit of great quantities of earthy matter in the form of intersecting and irregular plates, stretching downwards into the ventricle, as well as into the aorta, for an inch above the sinuses. In one of these cases the appearance of the opening might be aptly compared to that of the mouth of a shark in miniature; all traces of the valves had disappeared.

In these cases every superficial artery emitted a most distinct musical tone at each pulsation: the radial artery at the wrist, the palmar arteries, the ramifications of the temporal arteries, the anterior tibial, and the branches on the dorsum of the foot, all exhibited the same phenomenon. In two cases the sounds were distinctly audible to the patients, who were conscious of their existence at almost every point of the body. With one patient the perception of these sounds was the principal cause of his suffering, for his general health long continued excellent, and the heart's action was but little excited. This gentleman once observed to me, *that his entire body was one humming-top.* The loudness of the tone varied with the force of the heart. When I first saw him the sounds were audible at the distance of at least three feet; but when the force of the heart had been reduced by local treatment, the use of sedatives, and by removing all causes of bodily and mental excitement, the loudness of the sound at the aortic orifice was so much reduced as to render it inaudible, unless by applying the ear. Even under these circumstances the musical sound of the small arteries still continued, though not to such a degree as to cause annoyance to the patient. Dissection in this case showed but little disease in the aorta from about two inches above the orifice; the descending aorta and the arch were healthy; the left ventricle was hypertrophied and dilated; the general arterial system exhibited no disease.

Under such circumstances we may safely make the diagnosis of extensive and irregular ossification of the aortic orifice, with contraction, if the pulse be small and hard, and without contraction, if its ordinary volume be preserved.

To these cases, presenting physical signs sufficiently constant and well-marked to justify such a diagnosis of the condition of the valves as will be safe or practically useful, we may add the case of varicose aneurism, of which a description will be found in the section devoted to that subject.

But the practitioner must be prepared to meet with many cases which he will be unable to refer satisfactorily to any of these forms; for the complications of heart disease are so numerous and varied that, as we have said before, it becomes impossible to determine the exact nature of every case that may come before us. Fortunately it is unnecessary to do so, for if we can be certain that organic disease really exists, the treatment, as has been before remarked, will depend less on the nature of the valvular affection than on the vital and anatomical state of the heart itself.

Among the causes which concur to produce such varied phenomena in heart disease, the following may be enumerated:

1. The existence of valvular disease in more than one situation.
2. The changes incident to the advance of disease.
3. Alterations in the muscular structure of the heart.
4. Variation in the action of the heart.
5. Intercurrent attacks of endocarditis or of pericarditis.
6. Variations in the condition of the blood itself, causing the appearance and disappearance of anæmic, in addition to the organic murmurs.

To this catalogue other causes might be added; but the practical physician, knowing these things, will not feel that the difficulties of the subject reflect disgrace upon his art, when he considers that the great end of medicine is the proper treatment of the patient, rather than the exhibition of unnecessary refinement in diagnosis.

In connexion with this subject it is to be observed that many fall into the error of supposing that the loudness of the valvular murmur is proportioned to the extent of disease; and again, that murmur is so constantly associated with valvular disease, as that the absence of the former implies a freedom from the latter. But we know that in the arteries, at least, a very loud murmur may occur without any organic cause: and the existence of anæmic murmurs in the heart has been long recognised. We cannot then declare, that because a murmur is very distinct, the disease must be very considerable; nor can we, on the other hand, pronounce absolutely upon the healthy state of the valves merely because we can hear no murmur. This rule of course applies specially to those cases in which the symptoms, signs, and history lead us to suspect organic disease of some kind. We may lay it down as generally true that valvular murmur, once established as a consequence of valvular disease, continues, though showing occasional modifications, up to the period of death. But this is not always the case, and it is certain that the decrease of murmur may coincide with the increase of disease; and further, that in a case where at one time valvular disease was distinctly indicated by its proper murmur, this latter may wholly cease long before death, and when the organic affection has reached its greatest amount. This important fact is exemplified by the following case:

CASE XVI.—*Ossification and Contraction of the Mitral Valves; complete disappearance of murmur before death.*

A man past middle age was admitted into the Meath Hospital, labouring under the usual symptoms of disease of the heart, in connexion with chronic bronchitis and dilatation of the air-cells. He was affected with cough, dyspnœa, occasional orthopnœa, lividity of the countenance, and anasarca of the lower extremities. The action of the heart was much excited and irregular, with a corresponding pulse. A loud and permanent bellows murmur was heard in the region of the mitral valve; the second sound was healthy. Under treatment directed to relieve the lung, the urgent symptoms subsided, and he left the hospital, to all appearance convalescent, but still exhibiting the valvular murmur. Some months subsequently another attack supervened, and we

The various effects of organic disease on the function, structure, and form of the valves, is described in every work on pathological anatomy. In a practical point of view, it would be sufficient to recognise contraction or dilatation of the orifices, both of which conditions are attended by a permanently open state. This permanent patency is in some cases produced at an early period of the disease, while in others the valves may be so roughened by cartilaginous and ossific growths as to cause a murmur during the exit of the blood, while they yet remain competent to close the orifice. To this consideration we shall return, as it is one of those which may be indicated as opposed to over-refinement in diagnosis.

Valvular murmur is so much more frequently developed at the left than at the right side of the heart, that it is still a question whether we are in any case in a position to declare the existence of disease of the tricuspid or the pulmonary valves. If the relative position of the heart were always the same; if we had to deal only with cases of valvular disease, uncomplicated with change in the figure or volume of the heart; and lastly, if the rule were certain that the loudest sounds were to be found at the exact situation of the disease which produced them, it would be nearly as easy a matter to diagnosticate valvular disease at the right as the left side of the heart. But when we know that the investigator can seldom meet a case so circumstanced, and then reflect on the greater frequency of diseases of the left side, it becomes plain that the cautious physician ought not to commit himself hastily in a diagnosis of disease of the valves on the right side, much less declare its exact nature.

For it appears certain that we must be guided in our treatment of valvular disease less by the condition of the valves, than by that of the muscular portions of the heart. The practical physician, having satisfied himself that a valvular disease exists, will not devote too much time in ascertaining its exact nature; but he will examine into the vital and mechanical state of the heart's cavities. He will ascertain the amount of vigour of the heart; whether its force is above or below the natural standard; whether it is liable to excitement from slight causes; and whether irregularity of action or the opposite is its ordinary state. He will endeavour without murmur; yet at an early period of the affection well-marked murmur existed. In the next case we never observed murmur, and yet extreme valvular obstruction was found. It is probable that, had this patient been seen at an earlier period of the disease, the murmur would have been observed.

CASE XVII.—*Extreme Contraction of the Mitral Valve; Absence of murmur.*

A woman of middle age was admitted into my wards, labouring under aggravated symptoms of heart disease. The impulse was jerking and sudden, and the action of the heart intermitting and unequal. She suffered from cardiac anguish, want of sleep, and constant palpitation. The sound on percussion over the heart was clear, and both the first and second sounds were sharp and distinct, and totally without murmur. This observation I confirmed by many examinations, and under different states of the heart's action. On dissection the heart was found but little enlarged. The left ventricle was thickened and extremely firm, and the mitral valve so contracted that the orifice, which was irregular, could hardly admit an ordinary-sized quill.

It is now many years since the first of these cases occurred in the Meath Hospital, since which I have always taught in my clinical lectures that, with the advance of valvular disease, there might be a progressive diminution, and ultimately a complete cessation of murmur. It is to Mr. O'Ferrall, however, that we owe the publication of an important series of observations on this subject, in which he gives several well-observed cases illustrative of the disappearance of murmur in progressive valvular disease.[1] He believes, indeed, that with the advance of disease of the valve, the valve may be so altered as to prevent regurgitation, and that hence the regurgitating murmur disappears. On this point I shall not now offer any opinion, but refer the reader to Mr. O'Ferrall's memoir, which is one of great value. Explain it as we may, the great practical observation remains, that in certain cases of chronic valvular disease we may observe a diminution and ulti-

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[1] Clinical Researches in St. Vincent Hospital, by J. M. O'Ferrall, M. R. I. A., &c. Dublin Journal of Medical Science, First Series, vol. xxiii. 1843.

DISEASE OF THE AORTIC VALVES, WITH PERMANENT PATENCY.

The first sound unattended with murmur; the second replaced by a murmur which can be perceived to be double; this murmur is more or less audible along the course of the aorta, and, as regards the heart, is generally louder at the base than towards the apex.— The phenomena now described belong to disease of the aortic opening, and indicate that regurgitation into the ventricle takes place, owing to the defective condition of the valves. Such cases may be divided into two classes; those in which the disease is in an early stage, and those much more chronic, when the usual consequences of a hypertrophied and dilated ventricle have supervened. In the first case there may be no evidence of enlargement of the heart, and the characteristic visible bounding pulsations of the arteries may not be developed, or only seen in the neck. But in the more advanced periods we have, in addition to the loud double murmur at the aortic orifice propagated into the aorta and large arteries, the remarkable symptom of the visible pulsations of not only the great trunks, but of many of the smaller arteries which approach the surface. The radial pulse becomes quite characteristic. This is the jerking pulse, "the pulse of unfilled arteries" of Dr. Hope,[1] and we have no difficulty in recognising an enlargement of the left ventricle, if not of the entire heart. We owe the diagnosis of this disease to Dr. Corrigan.

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The action of the heart slow, feeble but generally regular, or only occasionally intermitting; a murmur with the first sound; the second sound healthy, yet a single murmur existing in the aorta and its large of independent observers. On this subject the following remarks of M. Forget are of great value.

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[1] Dr. Hope observes, that this character of pulse is produced by aortic regurgitation, in other cases as well as those where the reflux is into the left ventricle. He instances cases of communication with the pulmonary artery, or the mouth of the left ventricle. I have noticed this symptom in a case of true aneurism of the ascending aorta, in which the valves were competent to close the orifice. The name of collapsing pulse would be more appropriate, as the sensation given to the finger is that of a sudden disappearance of the arterial wave, which, as Dr. Corrigan has shown, is produced by the retrograde motion of a portion of the blood. See his original Memoir, Edinburgh Medical and Surgical Journal, April, 1832.

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"Is it true, as we hear it daily repeated, that the two hearts are situated, the one at the left and the other at the right side? So far as the cavities are concerned, such an arrangement exists but partially. It has been well observed by Bouillaud, Piorry, and others, that the right ventricle covers a portion of the left ventricle, before which it is thrown by means of the angular portion, whose summit corresponds to the orifice of the pulmonary artery.

"As to the auricles, the want of parallelism is still more evident. It is easy to perceive, in fact, that the left is thrown backwards and completely hidden by the common mass of the aorta and pulmonary artery; while the right, situated much more anteriorly, is projected towards the left; so that the right auricle alone is in contact with the sternum, to say nothing of the interposition of the anterior borders of the lungs.

"If we consider the relative position of the valvular orifices of the heart, we may strictly hold that the auriculo-ventricular openings occupy a left and right position, although the tricuspid orifice, like the cavity whose base it occupies, intrudes on the mitral to the extent of about a centimetre. Finally, the external angles of the auriculo-ventricular orifices stretch to the right and the left, but, in the case of the arterial openings, it is the reverse which occurs, for these orifices are exactly placed one above the other.

"These anatomical facts are manifest to all. How has it happened that they have been so long unrecognised, and that it is still imagined by observers that the cavities of the heart are regularly placed to the right and to the left, and that the right and left orifices are perfectly isolated?

"But this is not all, for the four orifices of the heart are so crossed, superimposed, and grouped, that their isolation is nearly impossible. The auriculo-ventricular orifices are only separated from the arterial openings by the thickness of the fibrous band surrounding the base of the ventricles, so that within a surface which could be covered with a five-franc piece, we find contained

Again, he observes, that when there is regurgitation through the valves of the pulmonary artery, a murmur accompanies the second sound; its nature and diagnosis are the same (the necessary inversions being made) as in the case of aortic regurgitation, except that the pulse is not jerking. A purring tremor has been found to attend dilatation of the pulmonary artery.[1]

It has been already observed that the practitioner should use great caution in giving a diagnosis, not only of the nature, but of the very existence of valvular disease at the right side of the heart; and Dr. Hope himself has dwelt on the necessity for the exercise of this caution, and pointed out that the signs he has specified must be perfectly well marked to justify the opinion. But although, in the last edition of his work, this excellent observer has not dwelt so strongly on the attainable certainty of special diagnosis in valvular disease, he still, I think, underrates the sources of difficulty that must accompany all attempts to discriminate the valvular diseases of the right side of the heart.

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as evidence of a recent and inflammatory affection. The same error is too often witnessed in the case now under consideration, and, as might be expected, the same disastrous consequences are found to follow.

The effects of injudiciously communicating to the patient that his heart is organically diseased, in conjunction with those of an ignorant and destructive medication, produce that very condition the absence of which has been the patient's chief safety. The heart becomes irritable, irregular, perhaps excited, and it is then no wonder that the *symptoms* of disease are superadded to the *signs*.

The recent development of the signs of a chronic, long preexisting disease is a circumstance which should be known to all who are concerned in the medical examinations for life insurance. Thus, it may happen, a life is passed as insurable after a careful examination. The insurance is effected, and yet in a short time the individual exhibits all those signs of morbus cordis which are supposed to indicate chronic disease. He may die of this disease within a few months after the completion of the insurance, and the payment of the sum insured be then contested on the ground that the disease was overlooked. I have known all the signs and symptoms of permanent patency of the aortic valves to occur within a few months after the effectuation of a large insurance, and yet at the period of the medical examination, which was made by one of the best observers in this or any other country, no sign of disease of the heart existed. In the same way I have known the signs of chronic mitral disease become most strongly developed in the course of a few days. These facts are of practical importance, for in the case of a judicial trial, on the ground of the incompetency or neglect of the medical examiner, many professional witnesses would incline to the opinion that the affection had been overlooked, rather than that it had become developed in so short a time after the examination. They would be influenced by the opinion that the development of disease and of its symptoms and signs are concurrent, a doctrine which we have seen to be untenable in acute, and, of course, far more so in chronic disease.

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In connexion with this subject it is to be observed that many fall into the error of supposing that the loudness of the valvular murmur is proportioned to the extent of disease; and again, that murmur is so constantly associated with valvular disease, as that the absence of the former implies a freedom from the latter. But we know that in the arteries, at least, a very loud murmur may occur without any organic cause: and the existence of anæmic murmurs in the heart has been long recognised. We cannot then declare, that because a murmur is very distinct, the disease must be very considerable; nor can we, on the other hand, pronounce absolutely upon the healthy state of the valves merely because we can hear no murmur. This rule of course applies specially to those cases in which the symptoms, signs, and history lead us to suspect organic disease of some kind. We may lay it down as generally true that valvular murmur, once established as a consequence of valvular disease, continues, though showing occasional modifications, up to the period of death. But this is not always the case, and it is certain that the decrease of murmur may coincide with the increase of disease; and further, that in a case where at one time valvular disease was distinctly indicated by its proper murmur, this latter may wholly cease long before death, and when the organic affection has reached its greatest amount. This important fact is exemplified by the following case:

CASE XVI.—*Ossification and Contraction of the Mitral Valves; complete disappearance of murmur before death.*

A man past middle age was admitted into the Meath Hospital, labouring under the usual symptoms of disease of the heart, in connexion with chronic bronchitis and dilatation of the air-cells. He was affected with cough, dyspnœa, occasional orthopnœa, lividity of the countenance, and anasarca of the lower extremities. The action of the heart was much excited and irregular, with a corresponding pulse. A loud and permanent bellows murmur was heard in the region of the mitral valve; the second sound was healthy. Under treatment directed to relieve the lung, the urgent symptoms subsided, and he left the hospital, to all appearance convalescent, but still exhibiting the valvular murmur. Some months subsequently another attack supervened, and we had a second opportunity of studying the case; and again he left the hospital, the condition of the heart remaining unchanged. We now lost sight of him for almost two years, when he again was admitted, labouring under his old symptoms, but in a very aggravated form. His strength had greatly given way, and the condition of the lung was such that death seemed imminent. The action of the heart was violent and distressing in the highest degree, but all valvular murmur had ceased, and never re-appeared. He was for a time relieved by treatment, but ultimately sunk under dyspnœa, after a protracted struggle. On dissection, the lung was found to exhibit the most extreme degree of emphysema, with sub-pleural vesicles and dilated tubes. The heart was large, red, and firm; both ventricles hypertrophied. The mitral opening was completely surrounded by a ring of bone. It was contracted, and exhibited no trace whatever of valves or tendinous chords. Viewed from the auricular side it presented a funnel-shaped opening, ending in the crescent-like slit described by Dr. Adams, while on the ventricular side it showed nothing but a glistening, white, bony ring, as smooth as polished ivory. Here, then, there were narrowing and induration of the orifice, and doubtless, also, free regurgitation; but yet the murmur which had existed in the earlier stages of the disease had totally disappeared. The subsidence, too, of this murmur was not to be explained by the weakness of the heart, for the left ventricle continued in vigorous action up to the time of his last agony, and its muscular structure was red and firm. Had this patient been seen by us only at the time of his last admission, no one would have thought of making a diagnosis of valvular disease. But the case is strongly illustrative of the principle, that where other circumstances indicate disease of the heart, the mere absence of murmur should not necessarily make us declare that the valves are healthy.[1]

In the case now given we observed great valvular disease without murmur; yet at an early period of the affection well-marked murmur existed. In the next case we never observed murmur, and yet extreme valvular obstruction was found. It is probable that, had this patient been seen at an earlier period of the disease, the murmur would have been observed.

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[1] The existence of the double sound of the heart in the latter period of this case, after the destruction of the valve and the cessation of the mitral murmur, is interesting, as bearing on the cause of the first sound, which here could only have proceeded from the ventricular systole, and the closing of the tricuspid valve. Dr. Hope attributes the first sound to the tension of the valve, and also to muscular contraction, but thinks that the latter has the smallest share in its production.

CASE XVII.—*Extreme Contraction of the Mitral Valve; Absence of murmur.*

A woman of middle age was admitted into my wards, labouring under aggravated symptoms of heart disease. The impulse was jerking and sudden, and the action of the heart intermitting and unequal. She suffered from cardiac anguish, want of sleep, and constant palpitation. The sound on percussion over the heart was clear, and both the first and second sounds were sharp and distinct, and totally without murmur. This observation I confirmed by many examinations, and under different states of the heart's action. On dissection the heart was found but little enlarged. The left ventricle was thickened and extremely firm, and the mitral valve so contracted that the orifice, which was irregular, could hardly admit an ordinary-sized quill.

It is now many years since the first of these cases occurred in the Meath Hospital, since which I have always taught in my clinical lectures that, with the advance of valvular disease, there might be a progressive diminution, and ultimately a complete cessation of murmur. It is to Mr. O'Ferrall, however, that we owe the publication of an important series of observations on this subject, in which he gives several well-observed cases illustrative of the disappearance of murmur in progressive valvular disease.[1] He believes, indeed, that with the advance of disease of the valve, the valve may be so altered as to prevent regurgitation, and that hence the regurgitating murmur disappears. On this point I shall not now offer any opinion, but refer the reader to Mr. O'Ferrall's memoir, which is one of great value. Explain it as we may, the great practical observation remains, that in certain cases of chronic valvular disease we may observe a diminution and ultimately a disappearance of the murmur, indicative not of any cure or diminution of the disease, but really of its increase. And, as has been well shown by Mr. O'Ferrall, such a diagnosis is not difficult when, coincident with or subsequent to the disappearance of the murmur, we find the continuance or increase of the ordinary symptoms of disease of the heart.

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[1] Clinical Researches in St. Vincent Hospital, by J. M. O'Ferrall, M. R. I. A., &c. Dublin Journal of Medical Science, First Series, vol. xxiii. 1843.

So much has been written on the differential diagnosis of the valvular diseases that, to many at least, the preceding sketch of the subject will appear meager and insufficient. But the great principle which is to be insisted on is, that the number of the special combinations of signs and symptoms which warrants a special diagnosis is but small. And, again, that in most examples of the second category, namely, those in which the differential diagnosis is doubtful or impossible, there will generally be no difficulty in determining not only that the disease is organic, but also what is the vital state of the heart, and the mechanical conditions of its cavities and its walls. We may, as has been shown, determine with sufficient accuracy three forms and seats of valvular disease, namely,

1. Disease of the mitral valve.
2. Disease of the aortic valve with permanent patency.
3. Disease of the aortic orifice without permanent patency.

But when it is asked—can we say whether the disease of the mitral valve is a narrowing or a dilatation, an ossification or a merely cartilaginous thickening?—we must answer in the negative. If we are asked—is the disease confined to a single valve?—we can, in many cases, give but a doubtful answer. If the question is raised—can we always determine whether the valvular disease affects the pulmonary or systemic portions of the heart?—the answer ought to be, that we have little but probability to guide us, for in any given case of valvular disease the chances that it exists at, or at all events predominates in, the left side, are very great. To distinguish, by referring to the points of greatest intensity of murmur, between the diseases of the valves on the right and left sides of the heart, cannot be safely done. This doctrine I have held and taught for many years, and as clinical observation advances we see its truth impressing itself on the minds of independent observers. On this subject the following remarks of M. Forget are of great value.

"Is it true, as we hear it daily repeated, that the two hearts are situated, the one at the left and the other at the right side? So far as the cavities are concerned, such an arrangement exists but partially. It has been well observed by Bouillaud, Piorry, and others, that the right ventricle covers a portion of the left ventricle, before which it is thrown by means of the angular portion, whose summit corresponds to the orifice of the pulmonary artery.

"As to the auricles, the want of parallelism is still more evident. It is easy to perceive, in fact, that the left is thrown backwards and completely hidden by the common mass of the aorta and pulmonary artery; while the right, situated much more anteriorly, is projected towards the left; so that the right auricle alone is in contact with the sternum, to say nothing of the interposition of the anterior borders of the lungs.

"If we consider the relative position of the valvular orifices of the heart, we may strictly hold that the auriculo-ventricular openings occupy a left and right position, although the tricuspid orifice, like the cavity whose base it occupies, intrudes on the mitral to the extent of about a centimetre. Finally, the external angles of the auriculo-ventricular orifices stretch to the right and the left, but, in the case of the arterial openings, it is the reverse which occurs, for these orifices are exactly placed one above the other.

"These anatomical facts are manifest to all. How has it happened that they have been so long unrecognised, and that it is still imagined by observers that the cavities of the heart are regularly placed to the right and to the left, and that the right and left orifices are perfectly isolated?

"But this is not all, for the four orifices of the heart are so crossed, superimposed, and grouped, that their isolation is nearly impossible. The auriculo-ventricular orifices are only separated from the arterial openings by the thickness of the fibrous band surrounding the base of the ventricles, so that within a surface which could be covered with a five-franc piece, we find contained and superimposed the two arterial openings, and the greater portion of the two auriculo-ventricular orifices."[1]

LATENCY OF CHRONIC VALVULAR DISEASE.

The doctrine that disease of the valves, when it is uncomplicated with any functional or organic lesion of the muscles of the heart, is often so latent as to be undiscoverable without physical examination, is one of the great truths for which we are indebted to the genius of Laennec. And it is not yet sufficiently insisted on, that valvular disease, even to an extreme degree, may affect the heart without there being anything in the previous history or existing symptoms which could lead us to suspect the existence of such a lesion.

A slow, and, as it were, silent, disorganizing process may be developed in one or more of the valves of the heart, without pain, without irregularity of action, without any circumstance which could awaken the attention of either the patient or physician; and thus years may pass by, the patient fulfilling without inconvenience all the duties of an anxious, active, and energetic life.

But, with the want of symptoms, there is, doubtless, for a period which is undefined, absence of physical signs as well, and though the disease is manifestly progressive, no murmur is established until the mechanical change has reached that point which is competent to produce acoustic signs attendant on the flow of blood through the altered orifice. Thus it often happens that we may, with great care, examine the heart and find no evidence of disease, yet in a short time, it may be in a few days, manifest physical signs are developed which indicate not a recent and acute disease, but an extremely slow and long existing affection, yet one which had not, until the period of the second examination, arrived at the point when it was at last attended with acoustic phenomena.

In the chapter on carditis I have dwelt on the error which is so commonly fallen into of considering a murmur which had existed for a long period, but was then for the first time observed, as evidence of a recent and inflammatory affection. The same error is too often witnessed in the case now under consideration, and, as might be expected, the same disastrous consequences are found to follow.

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[1] Précis des Maladies du Cœur, &c., par C. Forget. P. 7.

The effects of injudiciously communicating to the patient that his heart is organically diseased, in conjunction with those of an ignorant and destructive medication, produce that very condition the absence of which has been the patient's chief safety. The heart becomes irritable, irregular, perhaps excited, and it is then no wonder that the *symptoms* of disease are superadded to the *signs*.

The recent development of the signs of a chronic, long pre-existing disease is a circumstance which should be known to all who are concerned in the medical examinations for life insurance. Thus, it may happen, a life is passed as insurable after a careful examination. The insurance is effected, and yet in a short time the individual exhibits all those signs of morbus cordis which are supposed to indicate chronic disease. He may die of this disease within a few months after the completion of the insurance, and the payment of the sum insured be then contested on the ground that the disease was overlooked. I have known all the signs and symptoms of permanent patency of the aortic valves to occur within a few months after the effectuation of a large insurance, and yet at the period of the medical examination, which was made by one of the best observers in this or any other country, no sign of disease of the heart existed. In the same way I have known the signs of chronic mitral disease become most strongly developed in the course of a few days. These facts are of practical importance, for in the case of a judicial trial, on the ground of the incompetency or neglect of the medical examiner, many professional witnesses would incline to the opinion that the affection had been overlooked, rather than that it had become developed in so short a time after the examination. They would be influenced by the opinion that the development of disease and of its symptoms and signs are concurrent, a doctrine which we have seen to be untenable in acute, and, of course, far more so in chronic disease.

It is not impossible that in some cases physical signs may be developed at so early a period of chronic valvular disease that we may consider these signs as of little less duration than the organic change, but such a case appears to be an exceptional one. And in most instances a long process of progressive disorganization has in all probability been going on before the mechanical conditions of the parts are so altered as to cause distinct physical signs.[1]

We have already spoken of the error committed in taking chronic disease of the valves for an acute affection. This mistake is unhappily but too common, and in connexion with it two errors are generally made; first, in supposing the disease to be of recent origin; and next in believing it to be necessarily progressive. It would appear, however, that some valvular diseases, at all events, are either not progressive, or that they advance with such extreme slowness as to constitute a class of cases very different from the more common examples of these affections. Some of the evils which result from this error have been already pointed out, but the following case will illustrate the importance of these observations.

It is now many years since I was consulted by a gentleman under the following circumstances. The patient, after having enjoyed excellent health for several years, was attacked by an influenza, then epidemic, and in consequence of considerable bronchial irritation, consulted a physician. He did not complain of any symptoms referrible to the heart; but his medical attendant, while exploring the chest with a view of determining the amount of bronchitis, discovered a bellows-murmur masking the first sound of the heart at the left side. The patient was then informed that he laboured under disease of the valves of his heart, and the diagnosis was confirmed in consultation with some eminent members of the faculty. All his habits were immediately changed; he was accustomed to active exercise on horseback and on foot, and was in the habit of drinking wine freely, but all exercise was forbidden except slow walking on a level surface, while he was put on an extremely spare diet, and complete abstinence from fermented liquors was enjoined. This total change in his habits, coupled with the usual results of unnecessary medical treatment, and the apprehension of sudden death so unexpectedly brought before the mind of an ardent young man engaged in an active profession produced, as might have been anticipated, an extremely depressed condition of mind and body. It was under these circumstances that I first saw him. He was of a full habit; the pulse perfectly regular and of fair strength; and the heart's action tranquil. He assured me that he had never felt any palpitation or uneasiness about the heart until after the period when this murmur had been discovered; in other words, until after the time at which he had been forbidden to use stimulants or active exertion. I found a distinct, but not rough murmur with the first sound of the heart, confined to the region of the mitral valve; the lungs were healthy, and it appeared that he never had an attack of pulmonary congestion or irritation except that one for which he consulted the physician. Taking into account the previous good health and habits of this patient, and the fact that no symptoms of pericarditis or endocarditis had been observed in connexion with the attack of influenza, and also that his general health, and even the condition of his heart, appeared to have suffered by the change in his mode of living,—I suspected that this murmur was indicative of some very old, passive, and stationary valvular disease, and this suspicion was converted almost into a certainty by the patient informing me that seven or eight years previously he had suffered from a severe attack of rheumatic gout, which affected many of the joints. There could then be hardly a doubt that the murmur was established at that time, but that the diseased action had not been progressive; the valves had been mechanically altered, but not to such a degree as to interfere materially with their functions. So that we had in this case to deal with the cicatrix of a wound, as it were, rather than with the wound itself. I explained these views to the patient, and endeavoured to re-assure him as much as possible. He was advised not to give up his profession, and was allowed to use stimulants in moderation. Smoking was forbidden;

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[1] To use the apt illustration of a friend of mine, an American physician, who has been studying in Dublin during the present year, the case may be compared to that of the building of a tower at one side of a hill, the greater part of which must be completed before those on the other side are able to perceive its elevation above the horizon.

and I directed the patient to return to me within a year. He did so; I found him much improved in appearance and spirits, while the physical signs of the heart remained quite unchanged. I saw this gentleman once annually for several years. On the last occasion but one he had just returned from a shooting excursion in the highlands of Scotland, which had occupied nearly a month. During this time he was on foot, walking over mountains for eight hours a day, carrying a heavy gun and shot-pouch, and using a liberal allowance of diffusible stimuli, yet he never experienced any difficulty in respiration, and when I saw him he was in the highest state of health and spirits. It is now more than a year since I have seen this gentleman; he was then in perfect health, although the murmur continued unchanged.

That this individual has had a continued mitral murmur for upwards of twelve years, there cannot be any reasonable doubt, and the case is strongly illustrative of this principle in practice, —that we are not to confound the effects of a disease with the disease itself; and again, that we are not rashly to change the habits of living, as to exercise and the use of stimulants, in a patient who has been the subject of a chronic local disease, if we find that under the regimen in question, local disease has not been progressive, and that the general health has remained unimpaired.

Other cases might be adduced of the long continuance of murmur in the heart without any special symptom of disease, and we may even see men with a loud rasping murmur continuing for years, who are yet able to take violent exercise. I knew a gentleman who was advanced in life, and who had to my knowledge a loud and rough mitral murmur for four years, yet during each season he rarely missed a day's hunting, and was a bold and fearless rider.

Another case, in which the practitioner will do well to observe extreme caution in diagnosis and prognosis, is that of the combination of organic and anæmic murmurs. This combination is not unfrequent, especially in young females, and it is often difficult to say whether the organic or the functional disease has had the initiative. Under these circumstances we have generally, with the symptoms of anæmia, the physical sign of a mitral murmur unattended by evidence of hypertrophy of the heart. Who can say at the first, or even after many subsequent examinations, what is the actual condition of the heart in such a case? He would be rash indeed who would declare that there is no organic affection, especially when he reflects that the combination of an organic disease of the heart, sufficient to cause murmur, and of that state of the blood which produces the murmurs of anæmia, may not only arise, but is in all probability one of frequent occurrence. Of these observations the following case is an illustration.

Case XVIII.—A young girl, aged 18, presenting all the characteristics of anæmia and chlorosis, was under my care in the Meath Hospital in the year 1842. She presented the signs of organic disease of the mitral valves, but on taking her age and general condition into consideration, I suspended my diagnosis as to the actual state of the heart, and contented myself with endeavouring to improve the general condition of the patient. She subsequently came under the care of Dr. Bigger. She died in December, 1842, (having been altogether more than two years ill,) with symptoms of congestion of the lung and anasarca. On dissection the left auriculo-ventricular opening was found to be funnel-shaped, and so contracted as scarcely to admit the passage of a quill. The aorta and its valves were in a healthy state; the left auricle was distended and its parietes thickened.[1]

I adduce this case as an example of one of those in which the practical physician should abstain from a positive diagnosis as to the condition of the heart. When I saw the patient the physical signs were unquestionably those of organic disease of the mitral valves, but her age, anæmic condition, and the periodicity of her attacks, made me hesitate to declare what proportion of the phenomena was to be attributed to organic or to functional lesion.

In a communication which I made to the Pathological Society I mentioned the case of a lady, aged 20, who presented all the symptoms of the anæmic condition. She had violent palpitations after exercise, swelling of the feet, some lividity of the lips, and a loud musical murmur with the first sound of the heart. The second sound was healthy, but the loud musical murmur was audible in the aorta and its primary branches. There was no evidence of enlargement of the heart, and the lady was not hysterical. She was repeatedly seen by Dr. Chambers, Sir Philip Crampton, and myself, and the question as to the presence or absence of organic disease was never determined. She was, however, treated by chalybeates, tonics, and other measures calculated to improve the anæmic condition; and with this remarkable result, that all symptoms of chlorosis vanished, that the murmur left the arteries, that the symptoms of heart affection disappeared, so that she was able to ride, walk, and dance, with pleasure; but the mitral murmur never subsided, although it lost much of its musical character. She continued for three years to all appearance in perfect health, when, while in the act of leaving her father's door, on a visit of charity, she suddenly dropped dead.

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[1] See Transactions of the Pathological Society, Dublin, Jan. 1843.

This was manifestly a case of the combination of organic and anæmic murmurs, yet one in which a positive diagnosis was at first simply impossible. In speaking of anæmic murmurs generally, I shall return to this case, here only remarking that the difficulty which attended the diagnosis, at least in the earlier periods, did not interfere in any way with the proper, and as far as was possible, successful treatment of the patient. In this case, as the nature of the disease was doubtful, we held it right to give the patient the benefit of that doubt, and accordingly attention was directed more to the general than to the local state. We could not say whether the mitral murmur was wholly functional or partly organic, but we could recognise the anæmic condition from the general history of the patient, the scanty uterine action, and the arterial murmur while the second sound remained clear. To this condition, then, our treatment was directed; and it must be admitted by every one familiar with cardiac disease that the life of this admirable lady was prolonged by a treatment in which the organic disease was really neglected, and which, at least in the opinion of many, would have tended to its exasperation, for the remedies by which she regained her health were, iron, bark, wine, and active exercise, in conjunction with a full participation in all the enjoyments accessible to persons in her rank of life.

The preceding observations naturally lead us to inquire into the absence of symptoms as well as signs of confirmed affections of the heart. It will be found that this is not so unfrequent as might be supposed. A slow organic change of one or more orifices of the heart may go on without exciting any symptom which leads to the suspicion of disease; and the heart, by some power of adaptation, seems to adjust its action, so as to carry on the function of circulation without manifest disturbance. But on the occurrence of any general disturbance of the system, the signs and symptoms of a diseased heart are suddenly developed.

I exhibited in 1840, to the Pathological Society, the heart of a gentleman of middle age, which illustrated the above positions. The patient was a man of exceedingly active habits, who had up to his fatal illness enjoyed uninterrupted health. A few days before his death he was attacked with rigors, followed by symptoms of fever, attended with bronchial irritation. In this state he remained for two days, when he was seen by his physician, who found him labouring under fever, bronchial inflammation, and extraordinary excitement of the heart. The pulsations were exceedingly violent and tumultuous, and were diffused over a large portion of the chest. A bellows-murmur with the first sound attended these violent pulsations. For three or four days he went on tolerably well, when he expired suddenly. On dissection the brain was found healthy, but the heart exhibited some singular appearances. The left ventricle was distended to the last degree with fluid blood, and the aortic opening exhibited the most extreme amount of obstruction from ossific deposits that I have ever seen or read of. At first, indeed, it seemed as if there was no opening; but when examined on the ventricular side a very small slit was discoverable of about four lines in length and one in breadth, through which it was just possible to pass a fine probe.

Now this patient had never exhibited any symptom of heart affection up to the time of his fatal attack, nor had his medical attendant the slightest suspicion that chronic disease of the heart existed. Had this gentleman been presented for a life insurance, it is probable that, so far as his history and symptoms would go, he would have been passed as an excellent life. We cannot say that a physical examination of the heart would not have revealed this extraordinary disease, but it is quite possible that it would not have done so with this extreme degree of obstruction so long as the heart's action was tranquil. There might have been no murmur whatever, nor any valvular sound from the aorta; while the auriculo-ventricular and the pulmonary valves being healthy, there would have been two clear sounds in the heart. Again, from the extreme narrowing of the aortic orifice, the characteristic pulse of aortic patency would have been wanting.

Thus we have another case of the sudden development of the symptoms and signs of a chronic and long pre-existing disease; another illustration of the great fact, that the sufferings in disease, within certain limits at all events, are much less dependent on the mechanical than the vital condition of organs. Here there were no symptoms of heart disease till the fever of influenza set in, and then, the heart's action being disturbed, the organ became unable to carry on the circulation.

Another case of great aortic obstruction was brought forward by Dr. Graves. The bony matter filled the sinuses of the aortic valves, contracting the opening so that only a small quill could be passed. The patient was a gentleman, aged 54, of active habits; he had never felt any inconvenience nor any deviation from a state of health till about six months before his death, when, in walking up a hill, he was attacked with severe dyspnœa. He afterwards found that walking even on level ground produced great distress and a paroxysm of difficult breathing. After each attack, however, he seemed to be quite well. About a month before his death he was attacked with influenza, but he was not confined to bed; and after the disease had continued for a fortnight he consulted Dr. Graves, who found the heart beating violently and irregularly. A loud bellows murmur with the first sound was audible over the whole cardiac region, and it extended as high as the top of the sternum. He had bronchitis, with cough and asthmatic paroxysm. His symptoms progressed with great rapidity; complete orthopnœa set in. He became dropsical, and died rather suddenly.

The interest of this case consisted in the sudden development of the symptoms of a disease which must have been long in progress. Two causes concurred in inducing the change in the vital state of the heart which led to the fatal result: one, the over-exertion from walking up hill, and the other the attack of influenza.[1]

Although Bouillaud has suggested that under certain circumstances muscular fibre may be developed in the valves themselves,[2] we cannot as yet adopt his opinion, and therefore we must, in studying the general pathology of valvular disease, consider the valves as of a simple constitution, passive instruments, as it were, of the powerful and complex machine to which they are subservient.

It would be out of place, in a work of an essentially practical character, to enter minutely into either the anatomical or pathological nature of the different valvular diseases, particularly as abundant information on these points may be found in the writings of the German, British, and French investigators. But the question as to whether we are to consider all valvular diseases not only as arising from endocarditis, but actually as examples of this affection, in its acute or chronic form, has an important bearing on practice, and may fairly be examined in this place.

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[1] As illustrative of the effect of a general disturbing cause in developing the symptoms of a previously existing mechanical alteration of parts, I may allude to the case of a gentleman who was attacked with the symptoms of influenza, then epidemic, in a severe form. These having continued for three or four days, suddenly subsided, and he then, for the first time in his life, became affected with irritability of the bladder, so severe that he was forced to pass urine every five or ten minutes. The urine was perfectly healthy. These distressing symptoms continuing obstinate for a fortnight, an instrument was introduced, and a large calculus discovered in the bladder. The operation of lithotrity was performed with ultimate success.

[2] "Enfin, comme certaines parties du cœur de l'homme ont un développement beaucoup moindre que celles du cœur de bœuf, ce dernier peut nous montrer, avec des caractères bien tranchés, des élémens qui n'existaient pas, ou qui n'existaient du moins qu'à l'état rudimentaire dans le cœur de l'homme. C'ést ainsi, par example, qu'on trouve distinctement dans les valvules du cœur de bœuf des fibres musculaires, tandis qu'on n'en aperçoit aucun vestige dans les valvules du cœur de l'homme à l'état sain. Je dis à l'état sain seulement et non à l'état anormal, car il ne m'est pas démontré qû'a ce dernier état, il ne puisse se rencontrer quelques fibres musculaires dans les valvules. Je viens d'examiner, il y a quelques jours, le cœur d'un jeune homme fortement constituée chez lequel la valvule bicuspide était considérablement hypertrophiée. Or il y avait dans l'ébaisseur de cette valvule quelques fibres ou filets rougeâtres qui avaient une grande ressemblance avec des fibres musculaires très minces."—BOUILLAUD, *Maladies du Cœur*.

Considered with reference to practical medicine, we may divide cases of valvular disease into two classes, in one of which there is reason to believe that a carditis has been the first step in the morbid process, while in the second we are without evidence that the alteration of the valve has been in any way connected with an inflammatory process. And it is important to observe that even in the first class of cases, although the morbid process, originally set up by inflammation, may continue and produce successive changes, it does not follow that the inflammatory state persists, so that we should be often in error if we described even this class of cases as examples of chronic endocarditis. With the exception, perhaps, of the mere cohesion of the valves, the pathological changes which are observed are common to both classes. We meet in both thickening, opacities, atheromatous and earthy deposits, contraction and permanent patency; and there can be little doubt, even in those cases where progressive changes occur, that these alterations continue under the influence of processes very different from that of inflammation.

In pointing out one of the leading errors of the pathology of the school of Broussais, namely, that inflammation does not change its nature, we have alluded to this subject and showed the error into which practitioners so commonly fall, in continuing to treat as inflammatory a disease which has long since lost that character, or which, perhaps, never had it at all. We may apply these principles to the treatment of many other diseases of the heart, and especially to those of permanent insufficiency of the mitral and aortic valves. For it is hardly possible to overstate the amount of mischief done in many cases of chronic heart affections by practice founded not on experience, but on a false theory, which leads to the adoption of a general and local antiphlogistic treatment.

We are not, on the other hand, to believe that there are no circumstances in which we should treat a case of valvular disease as an inflammatory, and possibly curable affection. In cases of the appearance of a valvular murmur, in the course of or immediately after the subsidence of an attack of pericarditis, we are to use all proper means to remove the endocardial inflammation. So, also, in examples of the recent development of a valvular murmur in cases of excitement of the heart, even without pericarditis, the same practice is to be employed; and experience shows that in many of such cases the treatment is followed by success, and organic disease of the heart prevented. But we must be sure that the murmur is of recent origin, and we should take care not to prolong our treatment beyond a justifiable period. What that period may be, it is impossible to declare with exactness, for this must vary in each case, and the question of change or cessation of treatment is to be determined by the experience and judgment of the physician.

The persistence of the murmur for a week or ten days is regarded by Dr. Hope as indicating that the disease has passed into the chronic stage, and this he observes may continue for several weeks, or even months, and still be benefited by antiphlogistic treatment. I have seen several cases in which, after a month, there was this much evidence of a chronic inflammation, that stimulants seemed to over-excite the heart; but I think it probable that, should the murmur persist for more than three or four weeks, we should be very watchful, lest, by continuing a reducing treatment, we weaken the system too much in the vain endeavour to remove an organic change.

When we come to consider the treatment of chronic heart disease we may inquire how far, as in acute endocarditis, we may employ a tonic or stimulating treatment.

It is generally believed that, organic disease being once established, there is a progressive disorganizing process set up, which must end in death, either by rupture of the valves, organic disease of the remaining portions of the heart, or obstruction to the current of the blood. And this is true in the great majority of cases. But, as we have already seen, there is reason to believe either that this disorganizing process may be occasionally of singular slowness, so that the patient may live for many years in the enjoyment of good, or at least tolerable health, or that the diseased action is really arrested and the lesion becomes stationary. I have seen several cases which admitted of no other explanation. In these it is probable that, although to a certain degree altered in their anatomical condition, the valves still preserved their function, so that there was neither any notable obstruction or insufficiency produced. And thus, with a non-excitable heart, the patient was not only permitted to enjoy excellent health, but was even able for years together to lead an active life and make great exertions, while at the same time he used wine and a generous diet. It is in such cases that improper medical interference is followed by the worst results.

It may be stated generally that permanent patency, with or without contraction of the orifice, is the final result of chronic valvular disease. This is attended with various conditions of the cavities, such as hypertrophy, dilatation, or both these conditions combined. But we cannot lay down with any certainty what state of the cavities will be produced, or at least found at the termination of the case, for the changes in the muscular structure of the heart vary not only with the amount of obstruction, but with that of permanent patency; so that we may find in the state of the auricles and ventricles the effects not only of the valvular disease in its last, but those produced in its earlier stages.

The practical physician, on being called to a case of valvular disease, having satisfied himself of the existence of an organic change in the mitral or aortic valves, or, as it may be, in both, will then direct his attention to the following points, which are the important subjects for consideration. These are:

1. To determine whether there is much obstruction to the current of the blood.
2. To determine whether the permanent action of the heart is augmented or depressed.
3. Whether actual enlargement of the cavities of the organ has taken place.
4. Whether the action of the heart is regular or the contrary.
5. To ascertain, as nearly as possible, the duration of the disease.

For it is on these points that his treatment must turn, and his prognosis to a great degree be founded.

Thus, if he find that although there be a manifest murmur, say with the first sound, and in the region of the mitral valves, yet that there neither is nor has been any symptom of dropsy of the extremities; if the heart's impulse be natural, its action regular, the pulse corresponding in force and character to the action of the heart; the sound on percussion of the cardiac region natural; while the lungs show no sign of congestion, and the liver no evidence of enlargement, he will come to the conclusion that the case is one not requiring much interference; and he will be slow to alter any of the patient's habits if it appears that the murmur has continued with but little change for a length of time, and that the general health has not been impaired. He will, of course, so far as he can do so without exciting apprehension in the patient's mind, direct him to avoid whatever *experience in the particular case* has shown to over-excite the heart.

Again, if in a case of manifest valvular disease he finds that œdema of the extremities has occurred; that the patient has had attacks of cardiac asthma, or of hæmoptysis; that there is violent action of the heart, with a pulse small or weak; if the heart is acting irregularly, while percussion shows that its cavities are enlarged; and if it appears that attacks of cardiac suffering have been induced by various causes, such as over-exercise, hepatic derangement, mental anxiety, or the abuse of stimulants;—he comes to the conclusion that the cavities have suffered; that the disease is in all probability progressive; and his treatment and prognosis will be shaped accordingly, for he knows that he has to deal not only with a disease of the valves, but with its worst consequences; and that the chances of sudden death are much greater than in the preceding case. Finally, the physician may observe signs of a weakened heart. These are of two kinds, both characteristic.

1. An extremely irregular, weak, fluttering action, with a corresponding pulse, rapid, unequal, irregular, and intermitting. He will find it difficult or impossible to distinguish the first from the second sound of the heart.

2. A morbidly slow, but generally regular action of the heart; the impulse feeble or wanting, unless at periods of excitement, or when the patient is turned on the left side.

If, now, under either of these conditions he finds that the respiration is often suspended, or that the patient is affected with involuntary sighing, if there have been repeated attacks of syncope or pseudo-apoplexy, and that these symptoms are mitigated by the use of stimulants, he concludes that, with the valvular disease, which may be mitral or aortic, or both combined, there is a weakened state of the heart, and that in all probability the disease of fatty degeneration has been established. Common sense, to say nothing of medical experience, points out the treatment for such a case.

From what has now been said it will be seen that, while the diagnosis of valvular disease depends on the existence and appreciation of certain physical signs, the questions of prognosis and treatment depend upon the condition of the muscular portions of the heart. It is true, that in cases of confirmed valvular disease, there is danger of sudden death, generally from rupture of the valves or tendinous chords; but if we exclude the consideration of the state of the heart generally, we have no means whereby to judge of the probability of such an occurrence, for we cannot by any special acoustic character of the valvular signs determine what the exact anatomical change may be. Permanent patency, indeed, especially of the aortic valves, generally gives a characteristic double murmur, but if we exclude this case, we find that murmur attends a great variety of valvular disease, that it may be present in dilatation or contraction, in ossification, cartilaginous deposits, warty excrescences, perforations, adhesions, polypoid concretions, and aneurisms of the sinuses, and we might, perhaps, say with truth, that every variety of murmur may be met with in every variety of disease. As, however, so much depends on the condition of the cavities, and as these various diseases may exist with or without change in the muscular portions of the heart, we are justified in laying it down as a golden rule in practice, that in any case of valvular disease the determination of the condition of the auricles and ventricles is more important than that of the seat or nature of the valvular affection.

The question, as to why in one case the cavities remain unchanged in their mechanical and vital states, while in others such varied conditions of disease follow the valvular affection, is still undetermined. It may be that in those cases where the disease has sprung from an attack of carditis, the changes in the valve and the muscular portions of the heart proceed *pari passu*, so that we might be in error in attributing the dilatations and hypertrophy solely to the mechanical effect of the valvular disease. It may be, that the disease, by inducing an imperfect arterialization of blood, causes weakening of the heart, or that obstruction of the coronary arteries, as Dr. Quain has shown, may lead to the same result.

On the other hand, it appears certain that where a disorganizing process has commenced in the valves, independent of any inflammatory action, and advancing slowly even to the production of great ossific deposits, the cavities may for a long time remain free from disease. This will be more likely to occur in persons whose hearts are not excitable, whose digestive and respiratory functions continue good, and who have escaped the disturbing influence of officious medical interference, and the apprehensions resulting from being made aware that they are the subjects of incurable disorder.

Indeed the study of cardiac pathology leads irresistibly to the conclusion that in valvular disease the source of irregular and excited action is to be sought for less in the condition of the valves than in that of the heart itself. As there is no form of mere valvular disease which has not been found to occur with a perfectly regular action of the heart, we must look for the cause of irregularity and excitement in this affection to some other source; and it is to be borne in mind that the most remarkable cases of irregular action of the heart are those without any lesion of the valves. Gouty palpitation, hysterical or nervous affections, cardiac attacks depending on sympathy with the stomach or liver, and, lastly, the dilated and weakened condition of the heart, attended with pulmonary and hepatic congestion, as in the case of Mr. Colles, present the most striking examples, not only of irregularity, but of excited action; and these cases may occur independent of any valvular disease, or, if such exist, it is inconstant in its seat, nature, and amount, and incompetent to explain the condition in question. We too often find physicians giving an erroneous opinion from ignorance of these facts, for in their minds the ideas of irregular action and of valvular disease are so closely combined that they make the diagnosis of incurable disorder in cases where an emetic, an anti-nervous draught, the occurrence of gout in the extremities, or a few doses of a mercurial, will restore the natural action of the heart.

A remarkable case illustrative of what has been now said, occurred in Dublin some years ago. The patient, a lady of great intelligence, was for some years the subject of long continued attacks of violent and extraordinary palpitations, during which the action of the heart became greatly excited, extremely irregular, and attended by a loud bellows murmur, approaching to the *bruit de râpe.* During these attacks she was visited by several experienced physicians, who all concurred in the opinion that some extreme and singular disease of the valves existed. After having been the subject of this disease for several years, she consulted me. The paroxysm was then in its decline, after having lasted for some weeks, but the action of the heart was irregular, with a loud and somewhat metallic murmur apparently attending the first sound. She mentioned her anxiety that I should not make up my mind as to the nature of her case until I saw her a second time, which she arranged should be in the course of about ten days, observing that her physicians had not had fair play, inasmuch as they had only examined her heart during the continuance of its excitement. The patient was perfectly persuaded that she laboured under a fatal organic disease. I saw her again in about ten days; the heart's action was perfectly tranquil, the pulse natural, and every trace of murmur had disappeared. Several years afterwards I saw this lady; she was then in perfect health, and mentioned, with a good deal of self-complacence, that she had not only puzzled all her physicians, but had discovered her own cure, and this was in the use of an emetic at the commencement of each attack, a practice to which she had been led by the occurrence of accidental vomiting from the effect of some medicine which had been administered. She then determined to take an emetic of mustard or ipecacuanha on the supervention of each attack. The paroxysms became less and less severe, and finally disappeared. When I last saw her she was able to take active exercise, and the action and sounds of the heart were natural.

A case, probably of a similar nature, was that of a young man who was brought into hospital, suffering from extraordinary excitement of the heart, the action of which was so violent that the most severe form of carditis was believed to exist. The patient was treated with extreme but erroneous activity; he was repeatedly and largely bled, mercury was freely exhibited, and all other means of subduing local inflammation resorted to; yet not the slightest impression seemed to be made on the disease; and as his strength was much exhausted, while the action of the heart continued with terrific violence, the gentleman under whose care he was placed suspended treatment, the death of the patient being daily expected. A draught, containing ether, laudanum, and other ingredients, having been taken, was followed by full vomiting, after which the action of the heart became regular and tranquil; the murmur disappeared, and convalescence was rapid and complete.

DISEASES OF THE VALVES AT THE RIGHT SIDE OF THE HEART.

In the consideration of the question, as to how far we can determine the separate existence of valvular disease at the right side, or its co-existence with analogous affections at the left side of the heart, we may exclude cases of congenital malformation. Keeping in view the great object of clinical study, namely, the application of pathological anatomy to diagnosis and practice, we find that diseases of the valves of the pulmonary artery, and of the tricuspid valves, are rare as compared with the analogous affections of the left side of the heart. So great is this difference in frequency, that in practical medicine we may confine ourselves to the diseases of the mitral and aortic valves.

If, excluding anatomical considerations, it be asked, does our knowledge of clinical medicine justify a diagnosis of disease of the tricuspid or the pulmonary valves? the answer must be in the negative. This is at all events true with respect to the tricuspid valves, and as regards those of the pulmonary artery, it can only be said that, in the case of their permanent patency, we might expect that the to-and-fro murmur, similar to that in the analogous case of deficiency of the aortic valves, would occur, but wanting the accompanying phenomena of the aortic murmur and visible arterial pulsation. This condition was actually met with in the case communicated by Dr. Gordon to the Pathological Society of Dublin, to which we shall soon refer.

Such in fact is the diagnosis of permanent patency of the pulmonary valves given by Dr. Hope, who specially alludes to the absence of the "jerking pulse." But the case to which he refers is by no means satisfactory; and it is doubtful whether the murmurs really proceeded from the pulmonary valves. An attack of pericarditis, passing through the stages of effusion and absorption, occurred, from which the patient recovered and left the hospital with the supposed pulmonic murmurs still existing. We cannot admit the value of the remaining diagnostics given by Dr. Hope, namely, those which depend on the pitch or key of the murmur. Indeed this source of diagnosis must ever be fallacious, for the tone of all cardiac murmurs depends not only on the seat and nature of the disease, but also on the varying force of the heart. And, as we have already observed, the "jerking pulse" of Dr. Hope may be absent in the earlier stages of permanent patency of the aortic valves.

But although we cannot make a positive diagnosis of disease of the valves at the right side of the heart, yet this circumstance is not a source of embarrassment at the bed-side, for we know that such a lesion is rare, and even should physical signs exist, as laid down by Dr. Hope, they would indicate that which is of most importance to be known, namely, the organic nature of the disease. If we reflect that, rare as disease of the right valves may be, it is still more rare to find it uncomplicated with a similar affection at the left side, we need not concern ourselves as to the importance or difficulty of its special diagnosis.

A circumstance worthy of note, as showing the difficulty of determining the existence of disease in the tricuspid or pulmonary valves, is, that when the valves on either side of the heart are so affected as to give murmur, the normal sound of the opposite and corresponding valves is often so masked by that murmur as to become inaudible. If there be a mitral murmur, we lose the sound of the tricuspid valves, and if an aortic, that of the valves of the pulmonary artery. Reversing this, we find that the natural sounds of the left valves may be lost or modified, so that, in many cases of murmur, we are deprived of the advantage of comparing the healthy valvular sound on one side with the altered sound on the other. We have seen how doubtful all diagnostics drawn from the situation and tone of the murmur must be; and hence the element of probability on the one hand, and the association of symptoms and signs on the other, must be our chief guides in determining the seat of valvular disease.

As might be expected, no essential difference exists in the anatomical character of the diseases of the right valves as compared with those of the left, and the records of medicine give examples of the different forms of thickening, contraction, ossification, and cartilaginous growths in the sigmoid and tricuspid valves. It is laid down by authors, that the tendency to ossification is less seen in the disease of the right than of the left valves. Yet, though this is in all probability true, it remains to be determined whether we may not have been misled by the greater frequency of valvular disease at the left side of the heart.

"It is especially," says Laennec, "in cases of preternatural communication between the cavities of the heart, that the valves of the right side have been found affected." Bertin relates a case of this kind communicated to him by Louis (Obs. 67,) in which the tricuspid valve was partly ossified, and the sigmoid valves of the pulmonary artery formed a sort of fibrous ring hardly two lines and a half in width. In this case there was a small opening, two lines wide, between the right ventricle and the origin of the aorta. In another case observed by Bertin himself (Obs. 41) the foramen ovale was open, and the mouth of the pulmonary artery was "closed by a horizontal septum pierced by an opening two and a half lines in width." It appears probable that arterial blood has a great influence in predisposing to depositions of ossific matter, an opinion rendered still more probable by the consideration of the greater frequency of these ossifications in the valves of the left side of the heart.[1]

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[1] See the case of General Whepla, quoted by Louis in his "Memoire sur la Communicatione des Cavités droites avec les Cavités Gauches du Cœur," from the Journal de Medicine, vol. ix., p. 468.

Case XIX.—*Permanent patency of the Valves of the Pulmonary Artery; open Foramen Ovale; Double murmur at the base of the Heart not propagated into the Aorta; Absence of visible pulsation of the Arteries.*

For this important case I am indebted to Dr. Gordon. A boy, aged twelve, was admitted into the Hardwicke Hospital on the 1st of March, 1851, labouring under symptoms of severe pulmonary disease. The face was congested, and the surface cold, his pulse extremely feeble, and the expectoration copious and muco-purulent. A muco-crepitating rattle existed over the whole chest, and a remarkable thrill (*fremissement*) could be felt over the entire præcordial region. Along the sternum there was a well-marked double murmur, similar in every respect to that observed in the ordinary case of permanently open aortic valves. It was loudest at the base of the heart, and became less distinct as the stethoscope was moved towards the apex, in which situation, in fact, it ceased to be audible. There was, however, no visible pulsation in the carotids, subclavian, or radial arteries, nor any murmur or fremitus in those vessels. In the inter-scapular region the double murmur could be heard, although its intensity was greatly diminished.

This patient had been considered healthy until he had attained the age of seven years, when, after an attack of measles, he continued to suffer from cough, dyspnœa, and palpitation, increased by the least exertion. During his stay in hospital he was much relieved from the bronchitis, yet though the action of the heart became less violent, the fremitus and double murmur continued unaltered in extent and intensity.

In this case the existence of the purring thrill over so large a surface, taken in connexion with the anomalous circumstances of the case, led Dr. Gordon to make the diagnosis of an open foramen ovale.

On dissection, the heart was found but little enlarged, an oval opening, the longest diameter of which was about three-quarters of an inch, was found in the inter-auricular septum. The valves of the heart were generally healthy, with the exception of those of the pulmonary artery. These valves were thickened, shortened, and opaque, leaving a gaping orifice through which water passed freely when poured into the artery. This case is another illustration of the doctrine, that organic disease of the valves of the right side of the heart is most often met with when a preternatural communication exists between the systemic and pulmonary sides of the organ.

So far as this single case goes, it justifies the diagnosis of permanently patent valves of the pulmonary artery, which has been suggested rather than established by Dr. Hope and others, namely, that there should exist a double murmur at the base of the heart similar to that in aortic patency, yet without the propagation of murmur into the large vessels, or the throbbing and visible pulsation of the arteries.

On the subject of insufficiency of the valves of the pulmonary artery, Dr. Walshe observes, that "pulsation of the arteries would not accompany the double murmur of patency of the pulmonary valves." "By a singular fatality," he remarks, "while a certain number of examples of such destructive disease or insufficiency of the valves as must have led to full regurgitation have been observed *post mortem* in this country, in not one that I know of had the physical signs been clinically established. Theoretically the effects on the systemic and cerebral capillary circulation must be most serious, and a sensation of dyspnœa, arising from the smallness of the quantity of blood actually reaching the lungs by each systole might, unless the force of habit would counteract this influence, be expected."[1]

The case now given will supply to a certain degree the deficiency complained of by Dr. Walshe. But yet we cannot attribute the whole of the physical signs in this case to the permanent patency of the pulmonary valves, for the purring thrill may be considered to have arisen from the defective condition of the auricular septum. We do not usually find this sign connected with aortic patency, and it is hence unlikely that it would occur in the analogous condition of the pulmonary artery.

There is another form of insufficiency of the valves which arises not from disease of the valves themselves, but from dilatation of the cavities when carried beyond a certain point. It is probable that this condition will be found more frequently at the right side, where it may affect both orifices, and be attended with dilatation of the pulmonary artery. The case now given is illustrative not only of these conditions, but is one of those in which the grounds for a precise diagnosis were manifestly wanting, inasmuch as the physical signs might have been held to indicate a variety of organic lesions.

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[1] A Practical Treatise on Diseases of the Lungs and Heart. London, 1851.

CASE XX.—*Dilatation of all the Cavities of the Heart, of the Pulmonary Artery, and of the Aorta; Insufficiency of the Auriculo-Ventricular Valves on both sides; Fremitus over the Heart with a musical murmur attending the second sound; Replacement of the systolic sound on the left side by a soft murmur.*

J. Loughlin, aged 34, was admitted into my wards in November, 1847, labouring under general dropsy and symptoms of cardiac disease. This man had enjoyed good health for the last six years, and during that time had been temperate. About six months before admission he began to complain of cough and dyspnœa, attended with palpitation, which latter symptom occurred without any assignable cause. The dropsical condition commenced three months previously.

On admission his countenance was pale and expressive of great anxiety, and his whole appearance indicated congestion. General anasarca and ascites existed. The chest was clear on percussion, except in the region of the heart, which was dull to a much greater extent than natural. The jugular veins were distended and visibly pulsating. Bronchial râles were found over the chest. The heart's impulse was feeble, but it was attended with a most intense and extended fremitus, and a loud musical murmur synchronous with the second sound. To the left of the nipple a soft and indistinct murmur replaced the first sound. The radial pulse was very feeble, and beat 100 in the minute.

This patient sank rapidly. The heart was found to be enlarged to more than twice its natural volume; this increase of size was principally owing to dilatation of the cavities. Both auricles and the right ventricle were much enlarged. The left auricle was twice its natural size, with some hypertrophy. The aorta was of a bright red colour, and thickly studded with atheromatous concretions. It was dilated, but the orifice was quite perfect; the valves, though a little thickened, being competent to close the opening completely.

The right auriculo-ventricular opening admitted of five fingers being passed through it; its circumference measured six inches and a quarter. The valves were healthy, but evidently incompetent to close the orifice. The circumference of the pulmonary artery was not less than four inches; the valves healthy; four fingers could be passed through the left auriculo-ventricular opening; its valves were healthy, but its circumference measured five inches; the valves seemed insufficient to close the opening. The circumference of the aortic orifice was three inches and three quarters.

In this case the greatest amount of dilatation of the orifices seemed to be on the right side of the heart; thus,—

The pulmonary artery measured four inches in circumference; the right auriculo-ventricular opening, six inches and a quarter; the aortic opening, three inches and a quarter; and the left auriculo-ventricular, five inches.

We have in this singular case a combination of circumstances which would justify the withholding an opinion as to the exact nature of the disease. The murmur with the second sound, we know, is generally indicative of aortic patency, but the character of pulse was wanting. It was small and weak, instead of being large and jerking. And, again, the remarkable fremitus and the jugular dilatation indicated something in addition to disease of the aortic valves.

To analyze the phenomena in such a case, so as to determine which of them were owing to the dilatation of the pulmonary artery, and which to the enlargement of the auriculo-ventricular openings, would be impossible in the present state of our knowledge.

According to Dr. Hope, dilatation of the pulmonary artery is one of the rarest diseases incident to man. One case only, in which this disease was revealed by dissection, is given by Dr.

Hope, the circumference of the artery being nearly five inches. I believe, however, that dilatations of this vessel, though of less amount, are not unfrequent. I have, on several occasions, found dilatation of the artery in examples of Laennec's emphysema, and it is remarkable that no unusual phenomenon attended these cases.

We cannot, however, admit the rules for the diagnosis of this affection as laid down by Dr. Hope. Indeed, the whole of his statements on this point show the danger of attempting to establish rules for the exact diagnosis of the rarer diseases of the heart.

It may be asked, however, are we yet in a position to make the diagnosis of dilatation of the pulmonary artery. Here is a disease which is certainly one of much more rare occurrence than dilatation of the aorta. Can we, in a case where the dilatation is so great as to cause physical signs, certainly distinguish it from true aneurism of the aorta? I believe that in the present state of our knowledge we cannot safely make this diagnosis. I certainly would not venture to do so, even if the case presented all the signs given by Dr. Hope. So great is the variety in cases of aortic dilatation, that we cannot declare against the existence of disease in the aorta from the absence of any one of the signs of aneurism of that vessel, or the presence of any one of those which are supposed to indicate disease in the pulmonary artery. Thus aortic aneurism may cause a pulsation between the second and third left ribs with or without murmur. Large sacculated aneurisms of the arch of the aorta, too, may exist without external tumour or murmur, and without tremor, pulsation, or murmur, above either clavicle.

This case is illustrative of the principles already laid down as to the practical application of diagnosis. Here there was no difficulty in determining that the disease was organic, and that the heart was in a dilated and weakened state. The age and sex of the patient, the history of the case, the supervention of dropsy, all pointed out that a disease, not likely to be a merely nervous affection, existed. The feeble impulse, the pulsation and distention of the jugular veins, were indicative of a weakened heart with dilated right cavities, while the intense fremitus and musical murmur, although not propagated into the arteries, pointed out some important valvular lesion. So far, all that appeared useful to be known in this case was easily arrived at. The vital state of the heart was manifest, and the murmur obviously not an anæmic, not a nervous, but really an organic murmur.

But in such a case, to declare what was the exact cause of the fremitus and musical murmur with the second sound perceived over the whole heart, would have been to enter on a question incapable of solution, and one probably of no practical importance. We might have long and ingeniously speculated on whether they proceeded from disease of this or that valve, whether they indicated lesion on one or both sides of the heart, or whether there was any preternatural communication between the cavities,—without coming to any useful conclusion. Who could declare the exact state of the valves in this case, or say were they ossified, contracted, dilated, or permanently patent? Was there a dissecting aneurism, or did a coagulum interfere with the action of the heart? Why was the first sound on the left side replaced by a soft murmur, and what indications existed of the diseased condition of the pulmonary artery and the aorta?

If we consider that in chronic disease of the heart, when it is attended with symptoms and disturbance of action, with visceral congestion and dropsy, there is generally a complicated condition; that more than one set of valves is probably engaged, even though the physical signs seem to point out that but a single set are affected; and reflect that it is not always the more important lesion that causes the most prominent physical sign; and that the signs of disease on one side of the heart may mask the natural phenomena on the other,—we must be slow in giving a special or an exclusive diagnosis.[1]

DISEASES OF THE VALVES AT THE LEFT SIDE OF THE HEART.

It has been already observed that, if we consider valvular disease in relation to practical medicine, the affections of the left valves demand our principal attention. This arises from the following circumstances:

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[1] By exclusive diagnosis I mean that which declares, in an organic disease, that such and such portions of the heart are free from lesion, because none of the physical signs are present which usually attend the affections of these parts.

1. That they are so much more frequent.

2. That a certain proportion of them are in the first instance inflammatory, and therefore capable of being removed or controlled by medical treatment.

3. That they are liable to arise in the course of diseases which are of common occurrence.

4. That though, when established, they may exist for a great length of time without causing local or general disturbance, yet that they lead to disease of all the cavities of the heart, and give rise to special affections of the lung and brain.

5. That they more frequently terminate in sudden death than the affections of the right side.

It is easy to understand, when the complicated nature of the auriculo-ventricular valves is considered, which show an apparatus partly vital and partly mechanical, that an imperfect state of the valves may be induced by many causes besides inflammation. All the morbid processes that affect a serous structure by deposition, thickening, contraction, hypertrophy, atrophy, and transformation into an earthy or ossific state, may be found to cause imperfection of the valves. Again, whatever interferes with the action of the papillary muscles may impair that of the valves, as by over-action on one hand, and debility on the other. And again, the diseases of tendons by which, as Dr. Law has noticed, they are rendered brittle, probably bear a part in many cases of valvular disease. Lastly, coagula stretching through the orifices, and probably also purulent cysts, will impede the action of the valves.[1]

Thus, if we include inflammation, we have not less than twelve pathological conditions which may induce valvular lesion. And if the question be asked, can we in any given case, with the early history of which we are unacquainted, determine which of these causes has given rise to the disease, or how many of them are then concurrently producing it? the answer must be in the negative.

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[1] In the case of purulent cysts in the heart, consequent on phlebitic inflammation, which has been already given, one of the largest of the cysts was found behind the superior lamina of the mitral valve, which was, as it were, stretched over it, and rendered convex towards the ventricle. The specimen is preserved in the Museum of the Richmond Hospital.

The ultimate result of disease of the mitral valves is to destroy their mechanical function. And thus, from many causes, a permanently open state of the orifice is established. The period at which this change takes place will of course vary in different cases, but we find it with dilatation and with contraction of the opening. It appears more than probable that, when once this change in the mechanical state of the opening has occurred, that it remains permanent.

The views of Mr. O'Ferrall on this subject have been already alluded to.[1] In explaining the cessation of valvular murmur, while the organic disease continues, he advances the opinion that the regurgitation which had existed at the earlier periods, from the shortening of the valves, ceases in consequence of the contraction of the opening, so that their shortened laminæ become competent to close the diminished orifice. The order of phenomena would then be as follows:

1. Shortening of the mitral valves, causing regurgitation and murmur.
2. Contraction of the auriculo-ventricular orifice.
3. Cessation of regurgitation and of murmur, from the diminished orifice becoming adapted to the valves.

He observes: "If this, then, be the order in which the changes succeed to each other, is it not reasonable to suppose that shortening of the mitral valves most commonly anticipates the contraction of the orifice; and consequently that regurgitant disease in this part commonly precedes the phenomena of contraction?"

I have had no opportunity of observing the arrest of regurgitation under the conditions described by Mr. O'Ferrall, yet, without denying the possibility of such an occurrence, we must believe that the re-establishment of the function of the valves is not necessary to cause cessation of murmur in a case of progressive disease. It appears rather that it may occur in a contracted yet permanently open orifice, as in the cases I have detailed. And, so far as we know at present, the conditions capable of inducing this cessation of murmur are, smoothness of the edges of the orifice, attended with contraction.

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[1] See the Observations on the Nature of Valvular Diseases.

SYMPTOMS OF DISEASE OF THE MITRAL VALVES.

Although detailed accounts of the symptoms of mitral valve disease have been given by various writers, yet it is certain that the symptoms in question belong to complicated rather than to simple disease of the valves. And the complication is twofold, namely, that of a functional and an organic disease of the cavities of the heart. We know of no symptoms proper to mere disease of the mitral valves, and we have seen that these valves may have been long affected without any symptoms that could lead to a suspicion of disease. And in most cases, when the so called characteristic symptom of permanent irregularity of the heart is found, we may believe that organic change has taken place in the cavities of the organ; for an impulse which does not differ from that of health, a perfectly regular action, a pulse presenting nothing peculiar in its volume, rate, force, or rhythm, are commonly to be met with in cases where a distinct mitral murmur exists, and in which for many years the patient has shown no symptoms of disease of the heart, and has been able to use long-continued and fatiguing exercise.

In another set of cases we find this absence of symptoms, unless under the influence of fatigue or excitement, when increased action, palpitation, and dyspnœa, occur, but yet subside after a short period of time. And we may meet with cases of long-continued mitral murmur in which paroxysms of pain and cardiac distress are more likely to occur when the system is at rest than when the heart is excited. Such a condition may last for a great length of time, and with extensive and complicated disease, not only of the valves but of the cavities of the heart, as shown by continued mitral murmur and fremitus, and by the signs of enlarged cavities. The general health may be excellent, but the patient is liable to attacks of stinging pains in the region of the heart, which generally come on when the system is at rest, and are often absent during, and for some time subsequent to, the periods of active exertion.

We may safely hold that the symptoms of mitral valve disease, as laid down by authors, are those, not of simple change of the orifice, but of the complication of this state, with lesion of the muscular portions of the heart; and this, after all, is but repeating the doctrine of Laennec, which has been but scantily acknowledged even by the writers who adopt his views.

A contracted pulse, in cases where the orifice is narrowed, may be observed, but not with such constancy or character as to entitle the symptom to much consideration. And with respect to irregularity, experience shows that this condition is more intimately connected with lesion of the muscles than of the valves of the heart. In valvular disease, unattended by serious obstruction and uncomplicated with functional or organic lesion of the cavities, there is nothing which should cause irregularity of pulse. And it is probable that, were we to divide cases of valvular disease into two classes, namely, those with and those without irregularity, the latter would be found by far the more numerous.

There are, then, no special symptoms of disease of the mitral valves which distinguish it from other affections of the heart, for there is a class of symptoms common to almost all these affections. Nor can we admit that there are distinctive symptoms of valvular lesion of any kind, nor that, even when the disease is combined with hypertrophy and dilatation, irregularity of the heart is always present; for even under these circumstances the heart's action may be regular.

A violent impulse, while the pulse is small and weak, affords, according to Hope, one of the strongest indications of valvular disease. Yet these circumstances may occur in cases where no such lesion exists. They are met with in hypertrophy and dilatation of the right ventricle and auricle, in nervous affections, in anæmia, chlorosis, and occasionally in typhus fever.

Finally, we cannot declare the existence of disease of the valves from any character of the pain which may attend this lesion; and, even in the cases where it is present, no distinction has been observed between the pain in mitral, as compared with that in aortic valve disease.

But although pain of a decided nature, often severe and of a lancinating, pungent character, varying in its seat and extent in different patients, or at different times in the same person, sometimes stretching into the arm, as in angina pectoris, and at others singularly fugitive, and affecting successively various portions of the front of the chest, is a symptom of great importance and frequent occurrence in valvular disease; it is still to be determined whether it is indicative of simple valvular lesion, or of the combination with some form of hypertrophy. That it is more often met with in the latter case appears certain. I do not remember any instance of this cardiac pain where the disease was only to be discovered by auscultation, where the heart's action was tranquil, the pulse regular, and the signs of hypertrophy absent.

Nor is it yet determined what the nature of this pain may be, nor how far mere disease of the valves assists in its production. We may fairly doubt whether any real connexion, in the relation of cause and effect, exists between it and valvular disease at all; no matter whether we look on the latter affection in reference to its mechanical or vital effects. Dr. Hope believes that this pain is in general occasioned by the inelasticity of the ossified or otherwise indurated parts, which will not stretch equally with the other portions of the heart when the organ is labouring under palpitation or engorgement.[1]

If this opinion be well founded, we should expect that in any case in which these pains occurred, they would be induced by excitement of the heart. Yet it is not always so. And in certain cases we may not only see that the pains are not caused by active exercise, but that they are absent when the heart is unusually excited. I have long observed a case of this kind. The patient, when a child, was attacked with rheumatic fever and inflammation of the heart, in all probability an endo-pericarditis. On the subsidence of the fever, signs of confirmed valvular disease were established; it was at this time I first saw him; and since that period, now more than ten years ago, he has been under my care. He has grown up, and is a tall and powerfully developed man, although during the whole of this time the heart has exhibited manifest symptoms and signs of a great amount of valvular disease. This patient has also had repeated attacks of rheumatism, but of a mitigated character. The following conditions have been always present:

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[1] See Dr. Hope's Treatise, p. 356. The author observes that, "when inflammation of the interior of the heart exists it may occasion pain, but those authors have been unquestionably wrong who have considered inflammation to be the sole cause of pain, and have therefore assumed this symptom as a proof of the inflammatory nature of disease of the valves."

1. The impulse strong and extended, conveying the idea of a greatly enlarged heart; the pulse, however, not corresponding either in volume or force.

2. A purring thrill in the mammary region.

3. A loud and rough murmur with the first sound of the heart, having its greatest intensity to the left of the nipple, but heard over a large portion of the front of the chest.

4. The action of the arteries natural.

Now this patient has been for years liable to paroxysms of cardiac pain, of a well-marked and often distressing character, yet he has uniformly found that these pains came on when the action of his heart was most tranquil; and that whenever he suffered from excitement of the heart, induced by derangement of the digestive system or by the modified rheumatic attacks, he became free from pain. On many occasions, when warned against taking too violent horse-exercise, he has declared that the best mode of relieving the pains was to take a smart gallop on his horse and excite the heart into rapid action. It is difficult to explain these facts, if we attribute pain to the mechanical resistance of indurated valves; but it is more easy to reconcile them with the doctrine of engorgement spoken of by Hope, if we suppose that this engorgement was for the time lessened or removed by a more vigorous action of the heart.

Upon the whole, when we consider that pain of the heart is so commonly present without organic disease; that there are so many cases of long-continued valvular murmur, in which pain has been always absent; and lastly, that pain is in general so little associated with old mechanical changes of organs, and that it may occur in mere hypertrophy and dilatation of the heart;—the conclusion presses on us, that these cardiac pains are not necessarily connected with valvular disease, but are rather examples of some form of neuralgia, which may exist with or without organic disease of the heart.

The effect of mitral obstruction on the pulmonary circulation is twofold. It may produce a partial or general congestion, or an actual effusion of blood. In the first case we may observe the symptoms, and perhaps the signs of localized pulmonary apoplexy, while in the second, those of a more general congestion, with or without bronchitis, are noticed.

It appears probable that the production of a disease of the lung, answering to the description of Laennec's circumscribed pulmonary apoplexy, is the first and most common result of the valvular disease; while the second, namely, the general, though less intense congestion, is observed either during a paroxysm of cardiac asthma, or only towards the close of the case.

To explain the occurrence of isolated apoplectic effusions in the lungs of persons labouring under mitral obstruction is difficult. We find in various portions of the lung well-defined effusions of blood, of a size varying from that of a pea to that of a pullet's egg. Some have described this affection under the name of the nodular pulmonary apoplexy. In these cases, as distinguished from more general effusions, Hasse believes that the fluid is merely poured into the air-cells, without any rupture, while the adjacent texture remains healthy. This author states, that he found the whole of one lobe thus affected. Such an extent of disease, however, must be of rare occurrence.

But we would be in error if we supposed that this peculiar form of pulmonary apoplexy was dependent solely on mitral obstruction; for although the statements of authors, as to its connexion with disease of the heart, are not as accurate as could be wished, there is reason to believe that, while it may arise as a consequence of narrowing of the left auriculo-ventricular orifice, so also it may be produced, to use the words of Hasse, by hypertrophy of the right ventricle, causing an undue afflux of blood to the lung.[1]

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[1] Op. Cit. p. 247, Dr. Swaine's Translation. See also Allan Burn's Observations on Diseases of the Heart, 1809, as noticed in an important note by Dr. Forbes, in his translation of the work of Laennec, Art. Pulmonary Apoplexy.

Various affections of remote organs have been attributed to disease of the left auriculo-ventricular valves, which, however, it will be better to consider when we speak of the general effects of diseases of the heart.

From what has been now stated it appears, that we are unable by any study of symptoms alone, to determine the existence of mitral valve disease, either when it is uncomplicated, or when alterations of the cavities have occurred. In the first case, as we have seen, the disease may exist without symptoms at all, and in the second, those symptoms supposed to be characteristic are really not so, but are more or less common to many diseases of the heart.

PHYSICAL SIGNS OF DISEASE OF THE MITRAL VALVES.

We have already drawn in outline the character of these signs. The presence of a murmur which may be soft, hoarse, or musical, attending the systole of the heart, loudest towards the apex, and at the left side, and not propagated into the arterial trunks, is the chief indication of the disease. This murmur may be accompanied by a fremitus, and in many instances the second sound is unaffected.

In such a case, as has been already remarked, we might, taking other circumstances into consideration, make the diagnosis of organic disease of the mitral valves with a great degree of certainty.

But such examples as the foregoing are more often pictured in systematic works than met with at the bed-side; for here the observer who has taken books alone for his guides will meet with difficulties for which he is not prepared. A striking defect of many modern works on diseases of the heart is, that the authors assume not only that each disease of the heart has its special phenomena, but that no difficulty attends the determination of those accompanying circumstances, by which the seat of the abnormal signs is to be settled. The real difficulties of the subject have not been fully stated, and hence one cause of the differences of

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Dr. Townsend's Observations in the Cyclopædia of Practical Medicine, vol. i. p. 128, may be consulted. Drs. Hope and Walshe concur in attributing the nodular pulmonary apoplexy, in most cases, to disease of the mitral orifice.

opinion as to the exact nature of a particular case. It happens fortunately, that if the general diagnosis of organic disease be correct, the special diagnosis is of little value. This point has been already insisted on.

But to return to the subject in hand. We read that a murmur with the first sound, under certain circumstances, indicates lesion of the mitral valves. And again, that a murmur with the second sound has this or that value. All this may be very true, but is it always easy to determine which of the sounds is the first, and which the second? Every candid observer must answer this question in the negative. In certain cases of weakened hearts acting rapidly and irregularly, it is often scarcely possible to determine the point. Again, even where the pulsations of the heart are not much increased in rapidity, it sometimes, when a loud murmur exists, becomes difficult to say with which sound the murmur is associated. The murmur may mask not only the sound with which it is properly synchronous, but also that with which it has no connexion; so that in some cases even of regularly acting hearts, with a distinct systolic impulse, and the back-stroke of the second sound, nothing is to be heard but one loud murmur.

So great is the difficulty in some cases, that we cannot resist altering our opinions from day to day, as to which is the first, and which the second sound.

Again, many of the rules laid down for differential diagnosis depend on the transmission or non-transmission of the valvular sounds into the aorta. But this question, which, as discussed in books, seems of easy solution, is often, in reality, difficult to decide. For in many cases of mitral valve disease the murmur is found to extend along the sternum, and under both clavicles. Under these circumstances, although by ascertaining the point of maximum loudness to be towards the apex and to the left side, we may infer that the murmur extending over the chest is probably the mitral sound modified by distance, yet who can say that there is really no murmur in the aorta, especially when we know that disease of the aortic opening may exist, and yet the second sound remain unaffected?

But does the state of our knowledge of the signs of cardiac disease, and of vital acoustics in general, justify us in making an absolutely positive diagnosis, not only of the seat of the murmur, but of the nature of the disease, and the caliber of the orifice? This question must be answered in the negative, and we must receive as unproved and calculated to throw discredit on the science of diagnosis, all those rules and descriptions of special phenomena, supposed to apply not only to almost every pathological change of the valves, but every possible combination of these changes. In the ordinary cases of mitral murmur we cannot say whether the murmur is "constrictive" or "regurgitant," or constrictive and regurgitant; and we must reject a large proportion of descriptions of phenomena which, although the changes they are supposed to indicate be familiar to anatomists, are themselves of doubtful value. To the inexperienced the detailed descriptions of such phenomena as the intensification of the sounds of the pulmonary valves,[1] of constrictive murmurs as distinguished from non-constrictive, of associations of different murmurs at the opposite sides of the heart; of pre-systolic and post-systolic, prediastolic and post-diastolic murmurs, act injuriously; first, by conveying the idea that the separate existence of these phenomena is certain; and that their diagnostic value is established; and secondly, by diverting attention from the great object, which —it cannot be too often repeated—is to ascertain if the murmur proceeds from an organic cause; and again, to determine the vital and physical state of the cavities of the heart.

On this subject Dr. Graves' observations are of great value. "The chief means," says this true physician, "of distinguishing which of the valves of the heart is diseased is derived from the supposed direction of the sound. This is by far the most useful diagnostic mark we possess, and by it we may often, but not always, distinguish disease of the right from disease of the left side of the heart, and we may even occasionally, though not often, distinguish diseases of the auriculo-ventricular from those of the semilunar valves. Another means of diagnosis much relied on is taken from the morbid sound accompanying, and, therefore, being a perversion of the first or of the second sound of the heart; but as at each motion of the heart, valves are opened and valves are closed, a morbid sound may be produced by any change of structure which permanently prevents the complete opening or shutting of the valves; and consequently the same sound may arise either from changes of structure obstructing the advancing blood, or from changes permitting regurgitation;—in other words, it is impossible to judge at the moment a sound occurs, which of these is its cause."[1]

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[1] This is one of the signs noticed by Skoda as indicating constriction of the mitral opening, and giving a diagnostic between this affection and simple roughening of the auricular face of the valve. This doctrine, for the reasons already specified, cannot be received, and it has never happened to me to observe any augmentation of the second sound in cases of mitral murmur. Dr. Walshe observes, that "the least reflection on the unfrequency of direct mitral murmur, and on its frequent accompaniment, when present, by regurgitant mitral disease, will show how hazardous the assertion of Skoda is."—*Practical Treatise on the Diseases of the Lungs and Heart*, p. 226.

A case is given by Dr. Graves, which, however we may interpret it, is a good illustration of the accidents, so to speak, which may be in store for those who are over-confident in special diagnosis. A man of intemperate habits had for eight years laboured under palpitation and dyspnœa. When admitted into hospital he was emaciated and dropsical; pulse 94, regular; and there was no visible pulsation, thrill, or bellows murmur in the arteries of the neck or upper extremities.

The right side of the chest was dull, with weak and crepitating respiratory murmur. Loud respiration, free from any *râle*, was heard over the left side, which was clear on percussion. The impulse of the heart was strong and rather diffused; the sounds loud, the first being accompanied by a bellows murmur audible all over the cardiac region, but remarkably intense to the left of the nipple. This did not ascend along the course of the aorta, nor was it accompanied by any fremitus.

This patient remained for five weeks under observation, when he sank, no change having taken place in the physical signs of the heart. The right lung was found studded with tubercle, the left was healthy. The heart was hypertrophied, and the pericardium universally adherent, the union being effected by a dense cellular membrane. There was not the slightest trace of recently deposited lymph. *All the valves of the heart were perfectly healthy.*

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[1] Clinical Medicine, p. 922.

The ascending portion of the aorta was dilated, and its inner surface rough and scabrous from an abundant deposition of earthy matter. The arch and descending aorta were healthy, and the aortic valves perfect.

Dr. Graves inquires how could such a case as this be distinguished from one of mitral valve disease? and compares it with an example of mitral contraction given by Dr. Budd,[1] in which the physical signs were nearly identical.

Although difficult of explanation, this case is one of great value, as showing the necessity of caution even when the best marked signs of local disease may exist. It does not, however, appear certain that the cause of the murmur was the diseased state of the aorta, for it is difficult to understand why a murmur thus produced should not be propagated in the course of the current of blood, while in the opposite direction it was loudly audible. The murmur, though not produced by valvular disease, may have arisen from other causes, perhaps some alteration of the form of the ventricle consequent on the adhesion of the pericardium, perhaps also from the state of the blood giving rise to an anæmic murmur in the heart.

COMBINATIONS OF DISEASE OF THE MITRAL VALVES.

Of these by far the most common is disease of the aortic valves. In this combination the relative amount of each affection varies considerably. Thus, in a case recorded by Dr. Law,[2] the disease of the mitral valves did not go beyond a slight thickening of their margins, while the aortic valves were greatly altered, so as to render the opening permanently patent. Two of the valves were thickened, and the margin of the third turned towards the ventricle so as to resemble the state of the lower lid in cases of ectropium. The apex of the heart was formed principally by the left ventricle.

Another case of this combination has been recorded by Dr. Law. The patient, twenty-four years of age, had led an irregular life, and was attacked with spitting of blood, dyspnœa, and cough, which led to the supposition that he was labouring under phthisis. A mucous rattle existed over the chest. The impulse of the heart was considerable, a double bellows sound was audible at the lower part of the sternum, and a single murmur at the left mammary region. He left the hospital, but was re-admitted in the following condition. He appeared stupid and listless; his face was flushed, and the temporal arteries were throbbing, yet there was less action of the heart than previously, and the abnormal sounds were no longer audible. He replied but slowly to questions; he was partially paralyzed on one side; and had convulsive fits during the night previous to his admission. After the lapse of about ten days he suddenly became comatose, and died almost immediately.

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[1] Clinical Remarks at King's College Hospital. Medical Gazette, January 7, 1842.

[2] Transactions of the Pathological Society of Dublin.

The heart exhibited a double lesion, the aortic and mitral valves being diseased. The natural form of the heart was altered, its apex having become rounded. A quantity of grayish purulent matter was found covering the inferior surface of the brain. The left corpus striatum was softened, as was also the adjoining cerebral substance.

The diseased condition of the brain in this case was considered by Dr. Law to have arisen from defective arterial supply, and to this subject we shall hereafter return. Considered with reference to the physical diagnosis of valvular disease, it is to be noted, that two distinct kinds of murmur were observed, differing both in nature and seat; one, a double murmur heard at the inferior sternal region; the other, a single murmur evident to the left of the nipple. The first of these was obviously the murmur of the permanently patent aortic opening, and the second indicative of disease of the mitral valves. That such a combination would justify the diagnosis of the double lesion appears pretty certain, and we shall just now record a case which occurred lately in the Meath Hospital, confirming the diagnosis as given by Dr. Law.

But we must not expect to find both mitral and aortic murmurs in these cases of combination, for it may be, that from excess of disease, as has been formerly explained, the mitral orifice becomes so altered as to give no murmur during the passage of the blood. This probably occurred in the following important case, for which I am indebted to Dr. Adams.

CASE XXI.—*Contraction of the Mitral and Aortic openings; Thickening and Dilatation of the left Ventricle and Auricle, the lining membrane of the latter being thickened and opaque; Great dilatation of the pulmonary veins; Occlusion of the contracted Mitral orifice by a Coagulum.*

A gentleman, aged 40, had, fifteen years previous to his death, suffered from an attack of rheumatic fever. His countenance gave no indication of his being the subject of disease of the heart. During the last six months of his life he found that riding on horseback, or ascending an eminence, induced dyspnœa, and he gradually became incapable of taking any exercise beyond that of very moderate walking. He had little or no cough. The mere exertion of dressing in the morning produced great exhaustion, a symptom frequently observed in such cases. The pulse at the wrist was weak and irregular, while the action of the heart was very strong, especially towards the apex, and its pulsations seemed more numerous than those of the radial artery. Percussion gave a dull sound over the anterior portion of the left side from the second rib downwards. The veins in the neck were not turgid, nor did they ever become so, nor were there any symptoms of dropsy or emaciation.

A bellows murmur, very distinct towards the apex of the heart, but also extending along the great vessels, could be heard; and the diagnosis arrived at was that there was contraction of the left auriculo-ventricular and the aortic orifices.

The patient laboured under a presentiment that his death would be sudden, and this was verified by the result. On the day before his death he appeared to be in excellent health; in the evening he took a walk with his children, and remained out till eleven at night. At three o'clock next morning he experienced a sensation of faintishness, and complained of feeling cold, and at daylight was found dead in his bed. His lips were livid, and the cause of death seemed to have been asphyxia.

The heart was found much enlarged, the left cavities being principally affected. The three orders of carneæ columnæ were much hypertrophied. Two large fleshy columns, as usual, occupied the lateral margins of the contracted mitral orifice. They were in close contact with the ventricular or under-surface of the valves. This under-surface was strengthened by the chordæ tendineæ, which were thrown much into relief. The aortic valves were hypertrophied, and presented a convex appearance towards the ventricle, as if they were distended, but showing a triangular opening in the centre; each side of this triangle was nearly a quarter of an inch in length; it exactly occupied the centre of the area of the aorta. Adhesion had taken place between the edges of the valves, and their margins were thickened and rounded.

The left auricle presented some remarkable appearances. It was much dilated and thickened, and the openings formed by the pulmonary veins were singularly enlarged. They were oval, and fully an inch in length and half an inch in breadth. The lining membrane was opaque and greatly thickened. The valvular orifice presented the appearance of a semilunar fissure: viewed from the auricle, its convex margin was forward, and its concave backward. This crescentic fissure was found completely closed by a coagulum of the size of a filbert. This was probably the immediate cause of death, closing the orifice like a bullet valve.[1]

The existence, in cases of contraction of the opening, of an isolated and probably moveable coagulum in the auricle, capable of causing death by a sudden occlusion of the orifice, has not, so far as I know, been noticed by any author except Dr. Adams. It is a most interesting and important fact. In another case recorded by Dr. Adams the coagulum was rounded, and exhibited concentric layers. Here the process of occlusion was probably more gradual, as the coagulum itself exhibited on its surface a perfect cast or mould of the contracted orifice.[2]

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[1] The heart was exhibited by Dr. Adams to the Pathological Society, on the 18th of January, 1845. (See the Transactions, Dublin Medical Journal.)

[2] "The cavities of the heart," says Dr. Adams, in his observations on disease of the mitral valves, "I have in general found filled with coagulated blood, which in some cases I have seen assume the appearance of the polypiform concretions which so much attracted the attention of the older pathologists. Most of these coagula had the appearance of recent formations, but my friend, Mr. M'Dowell, last winter found in the left auricle of a subject who died of the disease we are now considering, a ball as large as a pigeon's egg; it was formed of the fibrine of the blood, was very firm in its consistence, and of a figure perfectly spherical,

Another important feature in this case is the dilated condition of the pulmonary veins. These vessels were enlarged in every direction, so as to be at least double their natural dimensions. If the frequent occurrence of apoplectic effusions in the lungs of persons who have laboured under contraction of the mitral orifice be considered, the state of the veins in the case now given acquires an additional importance. For it seems not unlikely that pulmonary apoplexy may be of two kinds, one produced by increased action and over-loading of the arterial system of the lungs, as when the disease arises from hypertrophy of the right ventricle; and the other from distention of the pulmonary veins, when the passage of blood from the left auricle is obstructed; in the first case the masses are formed by the unarterialized blood; in the second, by the blood after it has passed into the capillaries of the pulmonary veins. Local collections of blood, probably caused by over-distention of vessels rather than by rupture, take place, having an analogy to those collections of the bile which we find disseminated through the liver when the biliary duct is obstructed. It may be a question whether the co-existence of an hypertrophied right ventricle is necessary for the production of these apoplectic masses in cases of mitral obstruction. On this subject my experience does not warrant any positive opinion, but I have seen the apoplectic state of the lungs in a case where, at all events, none of the usual signs or symptoms of hypertrophy of the right ventricle were observed.

The following case presents an example of disease affecting the mitral and aortic valves. It is the only one in which, guided by the observations of Dr. Law, we ventured to make the diagnosis of the double lesion.

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except that there was an oblong depression on it, which corresponded accurately to the form of the edges of the fissure by which the left auricle and ventricle communicated; small fossæ also, which must have been produced by the bony spiculæ, were seen upon its surface; from all which it was manifest it could not have been of recent formation. We examined this curious specimen of polypiform concretion too accurately to be deceived upon these points, and this, and the heart in which it was found, we have preserved."—*Cases of Disease of the Heart, &c., by Robert Adams, M. D., &c.* (Dublin Hospital Reports, vol. iv.)

This curious specimen is preserved in the Museum of the Carmichael School of Medicine.

CASE XXII.—*Permanent Patency of the Aortic orifice, with Contraction and Ossification of the Mitral valves; Dilatation, with Hypertrophy of all the cavities of the Heart; Double bellows murmur at the base of the Heart, with a single murmur masking both sounds towards the apex; Great enlargement of the right auriculo-ventricular opening.*

A man aged 35, of intemperate habits, was admitted into the Meath Hospital in December, 1851. He had enjoyed good health until about four months previously, when he experienced a severe attack of dyspnœa, which came on suddenly, and for the first time. This distress in breathing gradually increased; and about three weeks before admission he was attacked with cough and severe pains in both shoulders; his expectoration became mixed with blood, and symptoms of œdema and ascites showed themselves. On admission, the veins of the neck were turgid, the lips livid, and the face bloated. We found the action of the heart to be strong and irregular, with visible throbbing of the arteries of the neck and upper extremities; but the pulse at the wrist wanted the usual volume observed in cases of insufficiency of the aortic valves.

We could distinguish four seats of valvular murmur.

1. A double bellows murmur at the base of the heart, propagated into the aorta and subclavian arteries. The carotids did not present murmur, but gave a hard and, as it were, hammering pulsation.

2. A loud single murmur to the left of the nipple, evidently systolic.

3. A distinct *bruit de moulin* at the junction of the second and third right costal cartilages with the sternum.

4. A hoarse systolic murmur audible in the inter-scapular region.

We also observed that the right lobe of the liver was enlarged. Symptoms of progressive pneumonia of the right lung set in; under which he sank in less than a fortnight from the period of his admission. For a few days before death he complained much of the beating of the heart at the right side of the chest; and the throbbing of the neck and in the radial artery almost wholly disappeared.

On dissection, the right lung was found in a state of purulent infiltration (third stage of Laennec.) The left lung was healthy, and the pericardium contained about eight ounces of clear serum.

Both ventricles were hypertrophied and dilated; the right auricle was considerably enlarged, and the opening into the ventricle augmented to nearly double its usual dimensions. We found the left auriculo-ventricular valves presenting the usual appearance of ossific deposit in an early stage. They were thickened, shrivelled, and incapable of closing: the aortic valves, cribriform, and with their edges covered with vegetations, permitted free regurgitation.

After what has been said of the dangers of over refinement in diagnosis, it will not be supposed that in this particular combination of murmurs we may *declare that both sets of valves* are affected. The opinion in this case was given as it were experimentally, and it happened to prove correct; thus corroborating the diagnosis of the double lesion as given by Dr. Law. But we must still hold that the double disease may exist without the presence of such signs, and, conversely, that their existence may imply some other form or combination of lesions. There is one point in the case worthy of note, as being of greater value in the diagnosis of the double lesions than even the character and seat of murmur, and it is, that the pulse wanted the volume commonly seen in inadequacy of the aortic valves. It will probably be found that if, with the double murmur under the sternum, and the visible pulsation of arteries, the pulse is small and irregular, we may suspect that there is mitral contraction as well as a permanently patent state of the aortic valves.[1]

The diagnosis of double valvular lesion in this case was founded on the observations originally made by Dr. Law, that, in certain cases of the contraction or insufficiency of the aortic valves, with a contracted mitral orifice, he could distinguish two seats of murmur: one, towards the apex, a single murmur; and the other, which is double, loudest at the base of the heart, and propagated into the great vessels. But it is not in every case of this combination that we can make this diagnosis, for the mitral valves may be so altered as that no murmur whatever shall be produced during the passage of blood through them; and again, the murmur from the aortic opening may be so loud, and also so propagated downwards into the ventricle, as to obscure the mitral murmur, even should it exist.

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[1] The state of the left auricle and the pulmonary veins was not noted.

If the question as to the practicability of the negative diagnosis, with reference to either orifice, be raised, it appears probable, that where a mitral murmur is manifest, it will be easier to determine the absence of disease of the aortic valves than to declare the integrity of the mitral valves in a case of aortic patency. The experience of each succeeding day devoted to the study of diseases of the heart will make us less and less confident in pronouncing as to the absence of disease in any one orifice, although no physical sign of such a lesion exist, if there be manifest disease in another, or again, if there be symptoms of an organic affection of the heart.

I cannot offer any statistical statement with reference to the frequency of this combination, but we may with safety declare that it is one of common occurrence. Forget holds that cases in which the aortic valves alone are affected are less numerous than is generally supposed, and are about as frequent as those of isolated disease of the mitral opening; and again, that the simultaneous affection of the two sets of valves is as frequently met with as cases of the isolation of disease in either orifice.[1] This statement is probably not far from the truth, if we consider the mere occurrence of anatomical lesion rather than the actual amount of disease. It is, however, probable, that if we discard cases of slight alterations, insufficient to interfere with the action of the valves, it will be found that there are more cases of isolation of disease of the mitral than of the aortic orifice. Such at least is my present impression, drawn not only from the results of dissection, but from experience of cases, in which, without the signs of insufficiency of the aortic valves, those of mitral disease have continued for many years.

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[1] The statistical investigations on which these views of Forget are founded were published by him in his Études Cliniques more than six years ago. The number of cases observed was 29, and the proportions were as follow:—Isolated aortic cases, 9; isolated mitral cases, 10; combined cases, 10.—(Précis Théorique et Pratique des Maladies du Cœur, p. 157.)

CONTRACTION OF THE MITRAL VALVES.

It has been shown, that if disease of the mitral valves be considered independently of functional or organic change in the cavities, it appears so devoid of proper or distinctive symptoms as to be undiscoverable without the aid of physical examination. The period of this latent condition varies in different cases, and when at last the so-called symptoms are produced, they indicate combinations which may have preceded, but which in most instances have followed on the valvular obstruction or insufficiency.

Among the contributions to our knowledge upon this subject which have appeared since the time of Laennec, the researches of Dr. Adams are to be placed first in rank of importance, as they are in time of publication. His memoir, which appeared in 1827, may be held to mark a period midway between that of the discoverer of auscultation and of the investigators of the present time. In this memoir we find many observations which subsequent observers have without acknowledgment put forward as original. Thus we find the law, as Forget terms it, of the dilatation *a tergo*, indicated by Dr. Adams, where he shows the effect of mitral obstruction in causing enlargement not only of the left auricle but of the right ventricle. Again, the doctrines as to the pulsation in the jugular veins, synchronous with the ventricular systole, and the natural insufficiency of the tricuspid valves, are here fully developed; and the special modifications of the form of the heart, according to the predominance of disease in the auriculo-ventricular, or the aortic valves, are accurately described. Lastly, the mechanism and effects of the regurgitant diseases of the mitral valves are detailed and exemplified; and if aught were wanting to establish Dr. Adams' character as a philosophical observer, it is the dignified silence which he has maintained, while subsequent writers have laid claims to the discoveries of facts which he long before had announced. For there can be nothing more commendable than to avoid controversy when the object is to establish the mere priority of discovery, rather than the value and nature of a newly observed fact. In a science like medicine, which advances or has advanced less by the discovery of any great principle than by the accumulation of isolated facts, it matters little to the right thinking man who, having discovered a new truth, finds it afterwards claimed by another, if it be established, and made available for good.

If we bear in mind that the so-called symptoms of narrowing of the mitral valves are in reality those of a lesion of the cavities of the heart, combined with valvular change, we can see that the general group of symptoms of disease of the heart may be expected to arise in this affection, and by disease not only of the left but the right cavities. The following analysis of symptoms will place this matter in a clear point of view.

1. *General Symptoms.*—Palpitations; dyspnœa on exercise, occurring independently of pulmonary disease; cardiac pains.

2. *Symptoms referrible to disease of the left side of the heart.*—Irregularity, rapidity, feebleness, and diminished volume of the pulse; syncope; hæmoptysis; sudden death.

3. *Symptoms referrible to disease of the right side.*—Venous turgescence; pulmonary congestion; pulsation of the jugular veins; varying enlargement of the liver; anasarca; want of proportion between the strength, and perhaps the rapidity of the action of the heart and pulse.

It will not be supposed that any one of these symptoms belongs exclusively to the lesion under which it is classed. Thus hæmoptysis may occur either from increased action of the right ventricle, or obstruction at the mitral orifice, causing dilatation of the left auricle and pulmonary veins. Again, signs of affection of the brain may be observed to depend on deficient supply of arterial blood, as in syncope, or upon turgescence of the venous system, as in the coma and asphyxia in disease of the right side of the heart: but still, this general statement of the symptoms will help us to take a broader view of the nature and effects of a valvular disease which was at first but an isolated affection.

The symptoms of mitral obstruction are divisible into two classes; viz., those which result from mechanical impediment to the flow of blood, and those which indicate irregularity in the action of the heart. Among the former we may place,—

1. The evidences of congestion of the lungs, as shown by the

symptoms of cardiac asthma, bronchial disease, hæmoptysis, and œdema of the lung.

2. Evidences of obstruction at the right side of the heart, with its consequences, such as hepatic and cerebral congestion, general dropsy, and venous turgescence.

The second class of symptoms, or those indicating disturbance of the action of the heart, are,—

1. Irregularity and often rapidity of action, which may be either constant, or excited by various disturbing causes.

2. Want of proportion between the force of the impulse of the heart and that of the pulse in the arteries; the latter being often small and indistinct, while the former is strong and manifest.

3. Want of proportion between the rate of the manifest pulsations of the heart and of the pulse at the wrist; the former being often apparently more rapid than the latter.

With reference to the want of proportion between the heart and pulse, not only as to force but rapidity, Dr. Adams has the following observations:—

"First, when we recollect that the right ventricle is actively enlarged, and at the same time pushed forward towards the sternum by the dilated auricles above and behind it, and, moreover, that these three cavities just mentioned have a resistance to overcome at the left auriculo-ventricular aperture, we have no reason to be surprised at the vigorous pulse of the heart, to which the diminished left ventricle can contribute but little, as it is placed so much behind its usual situation. Secondly, the pulse in the arteries is small, weak, and irregular, and less frequent than that of the heart,—because the pulse of the former is the indication of the state of the left ventricle, which, as has been already mentioned, is reduced in size. And we can account for the irregularity of the pulse in the arteries, when we bring to mind that the left ventricle derives from the auricle above it a very precarious supply of blood, which is probably often inadequate to fill its cavity. Under such circumstances, the left ventricle may contract in unison with the right, but the stream it has to transmit will not be sufficient to distend the arteries, or make the pulsation sensible. At such a moment there is a total failure of the arterial pulse, while that of the heart (caused by the action of the right ventricle) is strong and vigorous; hence the phenomenon characteristic of this disease,—the occasional double pulse of the heart for the single pulse in the arteries."

In corroboration of this view of the want of proportion in rate between the pulsations of the heart and the radial artery, the following case is given:—

"A woman, who had for about a year laboured under the ordinary symptoms of valvular disease, with running attacks of dropsy, presented the following conditions:—The chest was well formed; the action of the heart was rapid, strong, and irregular, while the pulse at the wrist was weak and thready; and although its beat was for the most part synchronous with that of the heart, there were often two, three, or even five pulsations of the heart at a moment; then all pulsation was suspended in the arteries, and could not be felt by the finger placed accurately over the radial artery. The pulse counted here ranged at the rate of about 120 in the minute, and the beats of the heart during the same time exceeded by 10, 12, or 15, that number. I have never seen the pulsations in the jugular veins more evident than in this case; *and I ascertained that their beats corresponded accurately with every pulse of the heart, and even with those which were not felt in the arteries; moreover, when pressure was made on the exterior jugular veins, two or three inches above the clavicles, the veins became distended beneath this point during their pulsations, even more than when the pressure was omitted.*"[1]

The doctrine that it is by the influence of the blood, either in its quality or quantity, that the ventricle is stimulated to contract, is strengthened by this observation. Independent, however, of this consideration, we may admit, that the feebleness and want of volume of the pulse in such cases is not to be attributed, as some might suppose, to a weakened state of the left ventricle, first because we have no anatomical evidence that such a condition is commonly attendant on mitral obstruction; and next, because in cases of manifest weakening of the heart, as in fatty degeneration, and in the typhoid softening, where the action of the heart is regular, though often so depressed as that the first sound is inaudible, no such phenomenon as the heart acting more rapidly than the pulse has been observed.

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[1] Op. cit., p. 426. I have taken the liberty of giving the latter portion of this passage in Italics, in consequence of its great importance.

This explanation of the character of the pulse in mitral disease was given by Dr. Adams in 1827. In Dr. Hope's work, of which the first edition appeared in 1831, the author, speaking of disease of the mitral valves, says,—"The explanation of the pulses in question I conceive to be as follows:—In the case of contraction of the mitral orifice, the left ventricle, not being freely supplied with blood, is not stimulated to contract at the natural intervals with suitable energy and with equal degrees. In the case of regurgitation, the ventricle, having lost the resistance of the mitral valves, expends the force of its contraction in the retrograde as well as in the forward direction, and also expels into the aorta a diminished quantity of blood, whence the pulse is proportionably feeble and small; further, as the regurgitation disturbs the regularity of the supply to the ventricle, more or less of intermittence, irregularity, and inequality are sooner or later the result."

It is plain that, as regards the effects of the diminished supply of blood to the left ventricle, the views of Hope and Adams are the same, although the observations of the latter are not noticed by Dr. Hope.

But even in the second part of the explanation, Dr. Adams has priority of observation. In his comments on a case of contraction and patency of the mitral orifice, he observes that "the heart was of a peculiar form, owing to the greater capacity of the right side than the left. The pulmonary artery was unusually dilated; the aorta contracted; the left ventricle was diminished in size; the auricle a little dilated; the mitral valves were not half their ordinary depth; their borders were shrivelled and puckered up, as if a thread were drawn through them, and contained some spiculæ of bone; they were manifestly incompetent to do more than half guard the aperture of communication between the auricle and ventricle. This aperture was contracted, but was still large enough to admit easily the extremity of the index finger to the first joint, and it must have permitted the blood to pass without much difficulty from the auricle into the ventricle. In consequence of the shortening of the valve, it imperfectly covered the auriculo-ventricular opening, and too readily allowed of a reflux of blood into the left auricle during the contraction of the ventricle; hence the effect of the heart, instead of being, as it is in the natural state, expended in propelling onwards the blood through the aorta, was partly lost, because of the imperfect state of the valve admitting a regurgitation of some of the blood which was destined to pass into the aorta; the heart was therefore obliged to reiterate its beats, to compensate by its quickness for that small quantity of blood it was capable of forwarding at one contraction through the aorta."[1]

It must be admitted, that the real or apparent difference of rate between the impulse of the heart and that of the artery, as observed at the wrist, has not yet received sufficient investigation. As one of the symptoms of disease of the mitral valve, it is of great value; and a difference of not less than fifteen beats between the rate of the heart and pulse has been observed. Even a greater discrepancy may occur.

I have lately observed a case of mitral obstruction in which two distinct conditions of the heart's action are to be seen. In the one the action is comparatively tranquil and regular, and the mitral murmur is evident. In the other, the heart acts with great rapidity and irregularity, and the murmur becomes imperceptible or nearly so. In the latter condition there is a marked difference of rate between the pulse at the wrist and at the heart, so much so that, taking all the doubtful pulsations of the radial artery into account, there remains a difference of between 30 and 40 pulsations in favour of the heart. In making this observation every precaution to avoid error was taken, and I found that the best method of ascertaining the actual number of the heart's pulsations was by applying the stethoscope to the lower portion of the sternum, where the contractions of the right ventricle give a sound much more distinct than those of the left.

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[1] "In this organic change of the valvular apparatus at the left side of the heart," observes Dr. Adams, "by which a return of blood from the brain and lungs was impeded, we find the source of the quickness of the pulse, the vertigo, the dyspnœa, and the sudden termination of these cases."—*Dub. Hosp. Reports*, vol. iv. p. 422. The author gives two cases of contraction of the mitral opening, in both of which permanent rapidity of pulse was observed; and he remarks, that "In both he found the mitral valves and auriculo-ventricular opening in a state nearly similar, although the effects of this organic change were so dissimilar that, one patient having died of apoplexy, and the other in an epileptic fit, it would not be easy to assign any reason for these differences, nor to explain why the cases terminated so speedily. They are useful, however, in showing, that even in the first stage of this disease, life is very insecure; and the dissections present us with what we have not often an opportunity of seeing, namely, the change of the mitral valves which takes place when this disease is in what may be termed its first stage."—*Op. cit.*

If we now compare the action of the heart with that of the pulse at the wrist, confining our observations to the characters of strength, rapidity, and regularity, we may admit three groups of cases.

In the first, there is little, if any, disturbance of heart or pulse. The relation as to time between the stroke of the heart and of the pulse corresponds to that in the state of health; there may be no irregularity or intermission, and the force of the pulse appear unaffected. All this time a distinct mitral murmur is to be recognised; yet, with the exception of this sign, no evidence exists of disease of the heart.

In the second set of cases we observe, not only a want of proportion between the strength of the beats of the heart and pulse, —the former being much stronger than the latter,—but also a difference in the rate of pulsations, those of the heart exceeding those of the pulse by a number which may vary from 15 to 25 or 30. In this case the want of volume in the pulse is owing to the contraction of the auriculo-ventricular opening, causing a diminished supply of blood at each systole of the heart, and the organ has probably the globular shape produced by the enlargement of one ventricle, while the other remains unaffected, or even diminished in size.

Finally, of the third group we have a type in the case by Dr. Fleming, presently to be given, where the diminished volume of the pulse appeared to arise from free regurgitation into the auricle, while the left ventricle was in a state of hypertrophy with dilatation. It is still to be determined whether in such a case the want of proportion between the heart and the pulse as to the number of beats is to be met with.

The existence of a permanently rapid pulse, with or without irregularity, and occurring in an apyrexial state of the system, should lead us to infer that some disease of the heart was present. The chances that such a condition existed would be greatly increased if irregularity coincided with rapidity of pulse; and these probabilities would be converted into almost a certainty by the discovery of murmur with either sound of the heart, a murmur which was constantly present, or only evident when the heart's action was comparatively slow and tranquil. But what opinion should be given in a case of a permanently rapid pulse without pyrexia, without valvular murmur, or any evidence of obstruction in the pulmonary or hepatic systems? Such a case indeed, is rare, but it may occur, and the question will arise, whether the absence of valvular murmur implies absence of valvular disease; or whether the case is one of that class in which a murmur would be discoverable if the action of the heart was slow. On this question, I have only to remark, that I have never seen the masking of valvular murmur by a rapid action of the heart, in which there were both the want of valvular murmur and the absence of signs of pulmonary and venous congestion. So that the conclusion appears justifiable, that a merely rapid pulse, if it be isochronous with the heart, does not necessarily imply that the individual has cardiac disease, and more especially if murmur has never been present, and if the lungs and hepatic system have exhibited no sign of disease.

Pulsation of the Jugular Veins.—This striking symptom, held by Lancisi to indicate a dilated state of the right ventricle, has received an additional value through the researches of Dr. Adams, who has shown that it often occurs in mitral obstruction, so commonly a cause of dilatation of the right cavities. This pulsation, though not necessarily present in mitral valve disease, is found to be synchronous with the contraction of the ventricle, an observation of great importance, not only with reference to the signs of cardiac disease, but as bearing on the entire theory of the heart's action in a state of health.[1]

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[1] The pulsation of the jugular veins, the "venous pulse" of authors, to which Testa has given the name of the arteriosity of veins (Malattie del Cuore, vol. iii. cap. xvii.,) was noticed long before the time of Lancisi, although the doctrine of its connexion with disease of the heart belongs to the latter observer. It was described by Galen as occurring in a case of severe cephalalgia (*vide* Commentaries on Hippocrates, as quoted by Testa, vol. iii., cap. xvii.) Testa quotes from Zuliani, with reference to a case in which the pulsations of the veins of the arm resembled those of the artery: "*Chirurgus venam secaturus confunderetur metueretque.*" The same author quotes from Uccelli (Observ. iii.) as to a case observed

The pulsation of the jugular veins, when occurring in disease of the mitral valves, results from the regurgitation of blood from the right ventricle into the auricle, by which the current descending from the jugular veins is repelled into those vessels during the systole of the ventricle. Dr. Adams has observed, that, the pulsation in the jugular veins is synchronous even with those pulsations of the heart which are not perceptible in the arteries. The following passage from his memoir is important.

"Mr. Hunter, in his Treatise on the Blood, has remarked that the valves of the right side of the heart did not so completely close

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in the hospital of Brescia, in which there was manifest pulsation of the lateral portions of the neck. The right auricle was natural, while the right ventricle "unice dilatatus aliquantulum apparebat." It is remarkable that Testa, in alluding to these cases and to others, where the auricle, to use the words of Morgagni, *prohibente crusta interna sive cartilaginea, sive ossea, ipsaque hujus, aut parietum reliquorum duritie, contrahere se non poterat, sed rigida, et inflexilis in perpetua dilatatione permanebat,*" (Epist. Anat. xviii., Art. xii.) leads us to infer that the pulsation of the vein must be synchronous with the ventricular contraction. In these cases he observes, that the reflux of blood by the superior cava is solely owing to this, that the right ventricle receives a greater quantity than can be admitted into the pulmonary artery, from which he says, in consequence of some defect in the auriculo-ventricular valves, it happens that the same contraction of the ventricle which transmits the blood to the lung, returns at the same time some portion of it to the auricle, from which, but a moment before, it had passed; hence the blood returning a second time into the jugulars, and meeting there the current flowing towards the heart, causes their sudden distention. Op. cit. vol. iii. p. 379.) The case of venous pulsation recorded by M. Hombert, is well worthy of study. The patient, a lady of about thirty-five years of age, had suffered for upwards of fifteen years from attacks of asthma, attended with violent palpitation and pains in the thorax. When the palpitations were most severe, distinct pulsations could be perceived in the veins of the arm and the neck. The frequency of these pulsations was slightly different from that of the arteries, but corresponded exactly with the violent impulses of the heart itself. When the paroxysm was over, the pulsation of the veins ceased. On dissection the heart was found of twice its natural size, and as flabby as a bag of soft leather. The cavities were greatly distended, and the parietes of the heart very much thinned; in both the pulmonary artery and the aorta, polypi were found, whose roots were attached to the internal surface of the respective ventricles. The coagulum in the aorta having been removed, was found not less than two feet in length. The clot, for a length of six inches, was firm, red, and had the appearance of flesh. Hombert attributed the pulsation to reflux into the veins on each contraction of the heart, —"*L'on pourroit comparer ce repossement surnaturel du sang dans les veines au gonflement et au reboussement des eaux coulantes des Rivieres par les hautes marées*"—and he attributes the distention of the heart to the obstruction of the arteries by the coagula. (Histoire de l'Académie Royale des Sciences, Année MDCCIV. p. 161.)

the arterial and auricular openings as those of the left; but this circumstance, in my opinion, has not been sufficiently noticed, nor the influence that such a structure may have on the circulation in its natural or morbid state considered. I look upon this difference in the valves of the right and left side of the heart to be a natural provision to allow of a partial reflux into the right auricle, on those occasions when from any cause the passage of the blood through the arterial opening is retarded. Such a provision was absolutely necessary in the right or pulmonary ventricle, as various natural causes must momentarily retard the passage of blood through the lungs. Let us suppose the right ventricle to contract vigorously at such a crisis. Some part of the valvular apparatus (which is not very strong at this side) or the ventricle itself might give way, were there not some other course for the blood than through the pulmonary artery: in the natural state of the heart it is probable that there is constantly some little reflux into the right auricle during the contraction of its corresponding ventricle, as the valves readily admit it, but the great swelling of the jugular veins is only seen when extraordinary efforts are made, or when, from any enlargement of the right side of the heart, it is capable of containing a larger quantity of blood than it can readily transmit through the lungs, or the left receive; on these occasions it is that the pulsations in the jugular veins become evident; they are synchronous with the action of the heart, and can more readily take place when the right ventricle has been preternaturally dilated, as it is not likely that the valve will increase in size and breadth in proportion as the auriculo-ventricular aperture enlarges."

It is still to be determined whether the form of jugular pulsation we have now considered is only to be met with in contraction of the mitral valves; so little, however, is known of the diagnosis of a permanently patent, yet dilated mitral opening, that nothing definite can be stated on the subject. There appears, however, no reason why, if the pulmonary circulation suffered from such a condition, we should not observe a jugular pulsation in this disease as well as in that of narrowing of the orifice.[1]

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[1] The important memoirs of Mr. Thomas Wilkinson King, on the safety-valve function of the right ventricle of the human heart (Guy's Hospital Reports, Nos.

Three morbid phenomena are to be observed in the jugular veins in organic diseases of the heart, namely:—1. Dilatation without pulsation; 2. An undulatory action which may be looked on as an approach to pulsation; and 3. A well-marked reflux pulse, perceptible to the touch as well as to the eye, and in a few cases, attended by a faint murmur, but yet one which corresponds to each beat in the vein. The simple dilatation may be seen independent of any irregularity of form; but in some cases the vein exhibits a knotted appearance, giving the idea of the existence of septa, which cause a narrowing of the caliber of the vessel at various points.

Of these conditions, the pulsation of the vein is the most important, and was held by Lancisi to indicate an enlarged state of

iv. and xii.,) are worthy of the most careful study by every one interested in this part of the subject. To Dr. Adams, however, is due the credit, not only of developing the doctrine of the safety-valve function of the tricuspid valves, considered physiologically, but of showing the entire bearings of the subject in reference to disease; and we cannot assent to the statement of Mr. King, that Dr. Adams does not assign any cause for the regurgitation, unless it be dilatation of the auriculo-ventricular aperture. (*Vide* note to his first memoir, p. 126.) An examination of what Dr. Adams has said, not only with reference to the normal state of the tricuspid valves, but also when he compares them with the mitral valves, will establish what has been now advanced. We have above quoted his observations on the insufficiency of the right valves, considered as a natural provision. Farther on he says:—

"Before I conclude these observations on the healthy and deranged action of the auriculo-ventricular valves, I may remark, that the mitral valve so perfectly closes the aperture of communication between the left auricle and ventricle, that in the natural state no reflux whatever is admitted. This (the reflux,) so useful at the right side of the heart, would have been not only useless but injurious at the left side of the organ, as we find the general arterial system at all times equally ready to receive the blood during the systole of the left ventricle; and if the mitral valve did not perfectly close the left auriculo-ventricular aperture, a great deal of the force of the aortic ventricle would be wasted, whereby it would be incapable of moving the mass of blood which was destined to fill the arterial system. Pathologists, in looking to the different nature of the lining membrane at the two sides of the heart, as a means of explaining the greater liability of the left side to disease, have, perhaps, too much overlooked this circumstance, that while, from the unyielding nature of the mitral valve, all reflux into the auricle is prevented, from this very cause, which renders it effective in the circulation, is it exposed to more frequent injury from which organic disease may arise, and the ventricle to which it belongs become more liable to be ruptured by its own efforts."—*Dublin Hospital Reports*, vol. iv. page 489.

the right cavities of the heart. It is essentially a proof of obstruction to the pulmonary circulation and an overloaded state of the right ventricle. Hence we may infer, that it should occur in the following cases:—

1. Obstruction of the pulmonary artery or its valves.
2. Dilatation of the right cavities of the heart.
3. Obstruction of the left auriculo-ventricular opening.

It is not fully determined whether, in the last case, the sign in question indicates a permanent organic change of the right auricle and ventricle, such as dilatation with or without hypertrophy, or whether it may not arise from temporary distention of these cavities. In the case recorded by Hombert, it was remarked, that the pulsations in the jugular and brachial veins were most evident during the paroxysm of cardiac asthma.

Although the facts brought forward by Mr. King seem to establish that, in certain cases, a pulsation of the veins, independent of organic disease of the heart, and really propagated from the arteries through the capillary circulation, may be met with, yet, on the other hand, it appears certain, that the venous pulse is more frequently the result of regurgitation from the right ventricle. We owe to Dr. Benson one of the best recorded cases of venous pulsation, in which the veins on the back of the hand and the superficial veins of both upper extremities showed a distinct pulsation. The veins were prominent, and by some the pulsations could not only be seen but felt. These pulsations were a little later than those of the radial artery. In consequence of the increased action of the carotids it was difficult to say whether the jugular veins were pulsating. During each act of respiration they became distended, and then collapsed; whilst a confused, tremulous pulse incessantly agitated them.

In consequence of the rapid supervention of coma, no accurate history of this case could be obtained; but physical examination showed that the heart was hypertrophied, and that there existed some important valvular disease.

A small bleeding having been made from the arm, Dr. Benson was surprised to find that the blood did not come *per saltum*, although pulsation was observed in some of the veins below the bandage. The pulsation had ceased and remained absent for the following day; it returned, however, and remained for the next three days, and the patient sank. A small bleeding was performed on the day before death, when it was found that the blood flowed *per saltum;* from this time no motion in the veins could be seen.

"The heart," says Dr. Benson, "was at least twice the usual size. The auricular appendages, especially the left, were remarkably large. The right auricle was dilated and a little hypertrophied. At the posterior margin of the foramen ovale a particle of osseous matter was observed. The right auriculo-ventricular opening was very large and gaping. The right ventricle was dilated and hypertrophied. Its cavity was twice as large, and its walls twice as thick as usual. The floating margins of the tricuspid valves were thickened and studded with small cartilaginous nodules. The pulmonary artery was healthy, but its valves appeared somewhat thickened, and their corpora sesamoidea much developed. The left auricle was enlarged, its walls thickened, and the lining membrane peculiarly white and opaque. The opening from it into the ventricle was too small to admit the finger; it was an irregular slit-like opening, surrounded with cartilaginous and osseous deposits. The left ventricle was dilated, its walls a little thickened, but softer and paler than those of the right. The mitral valves contained calcareous and cartilaginous deposits. The aortic valves were greatly thickened, and filled with osseous matter. The aorta, too, had osseous deposits. The superior vena cava, the innominatæ, jugular, and subclavian veins, were slit up and carefully examined: nothing peculiar was observed in them; their coats were of the usual appearance, and their valves in the ordinary situations. The abdominal viscera were healthy. The brain was pale and bloodless: it showed no sign of congestion, nor of any disease except that the ventricles contained about half an ounce of clear serum."[1]

We must agree with Dr. Benson in his opinion that the pulsation in this case was regurgitant, and to be attributed to the condition of the right ventricle; and the case strongly corroborates the views of Dr. Adams as to the connexion between venous pulsation and the contraction of the mitral orifice, the remote cause of the disease in the right cavities of the heart.

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[1] A case of pulsation in the veins of the upper extremities, by Charles Benson, M. D. Dublin Journal of Medical Science, vol. viii. First Series, 1836.

But the occurrence of a venous pulse does not necessarily imply the existence of a chronic and incurable disease. I have noticed well-marked jugular pulsation in a case of acute pericarditis. The patient recovered, and there was no evidence of any organic disease prior to the inflammatory attack. In this case it is possible that the right ventricle was weakened and temporarily dilated, so that the tricuspid valves, naturally insufficient, were rendered still more inadequate.

In conclusion, it is to be observed that among the causes of cardiac asthma, contraction of the mitral opening is to be enumerated. It is not improbable, also, that a dilated orifice, with inadequate valves, may produce the same set of symptoms. We have, in various works, good descriptions of a paroxysm of cardiac asthma; but I do not know of any recorded observation of physical signs occurring in the paroxysm beyond those which relate to the excited and irregular action of the heart. The case I shall now detail will furnish some addition to our knowledge on this subject.

A girl of about eleven years of age, of delicate habit, has from a very early period of her life suffered from attacks of extraordinary dyspnœa and orthopnœa, in the intervals between which her health seems to have been good. She is of a delicate make and nervous habit, but is able to take active exercise; and no appearance of disease of the heart is shown by her ordinary mode of breathing or the expression of the countenance. There is, however, a permanent and slightly rough systolic murmur in the heart, loudest near to the left mamma.

In this case the paroxysm is liable to be excited by indigestion, by fatigue, or cold. I have had many opportunities of seeing this child in the intervals of her attacks; and on a late occasion I was called to see her in a fearful seizure of her disease. The pulse was small, unequal, and rapid to the last degree; and the respirations more accelerated than in any case I had ever before witnessed. There was constant cough, with wheezing, and an expression of dreadful anxiety. When I saw her she had been twelve hours ill, and I found that the left side of the chest was absolutely dull, except in the postero-inferior portion. It was as dull as if the lung had been compressed by a copious effusion. The heart's action was excited, and so irregular that to analyze the sounds was impossible. Considering that this child had not been a day ill, and also that the signs of dislocation of the heart were wanting, I felt great difficulty in determining from what cause this dulness proceeded. Treatment calculated to relieve the digestive system was adopted, and some anodyne and antispasmodic medicine given. These measures were followed by a considerable diminution of the excitement of the heart; the clavicle now became clear on percussion. During the next twenty-four hours the upper portion of the sternal region recovered its sound, and on the fourth day the dulness had completely subsided, and the chest had its natural sound, with distinct respiratory murmur. These observations I made with the greatest care, and on a careful consideration of all the circumstances, I can come to no other conclusion but that the dulness was produced by a sudden and extraordinary distention of the left auricle, so great as to displace the lung. Her recovery from this attack was much slower than usual, and several weeks elapsed before the heart was restored to its ordinary action. The paroxysm was not attended with cyanosis.

If we reflect on the causes which ordinarily produce complete dulness of the upper portions of the chest, we cannot find any which would explain the signs in this case. If it be recollected that this complete dulness was suddenly produced, probably within the course of a few hours; that there were no preceding symptoms or signs of consolidation from any cause; that the signs of pleural effusion were wanting, inasmuch as there was no displacement of the heart; that the postero-inferior portion of the side was clear; that the sonoriety of the chest was restored, although no antiphlogistic treatment was used; and finally, that no crepitus of resolution, or any friction sounds, attended the disappearance of dulness;—we cannot but believe that it was really caused by a temporary distention of the heart; and in all probability, reasoning from the symptoms then existing, and the ordinary state of the patient, that the left auricle was the seat of a vast accumulation of blood.

DISEASE OF THE MITRAL VALVES WITHOUT CONTRACTION.

The division of cases of mitral valve disease into the regurgitant and non-regurgitant forms cannot be maintained; for the effect of organic change in the valves must be, in most instances, to produce regurgitation to a greater or less degree. The following division appears more justifiable:—

1. Contraction of the orifice.
2. The diameter of the orifice remaining unchanged.
3. The orifice dilated.

Our knowledge as to the special or differential diagnosis of the two latter varieties is still limited; and little more can be said than that, as compared with the first or ordinary case of contraction of the mitral orifice, they often want the symptoms of mechanical obstruction, or at least that these are more slowly developed; and again, that when the orifice is dilated, we may have symptoms analogous to those of a weakened heart.

The following case occurred under the care of Dr. Fleming, to whose kindness I am indebted not only for the report, but for an opportunity of seeing the patient.

CASE XXIII.—*Sudden development of symptoms of organic Disease of the Heart; Repetition of Pseudo-apoplectic attacks, attended by ephemeral Hemiplegia and Jaundice; Dilatation of the left ventricle and auricle; Great enlargement of the Mitral orifice.*

A gentleman, aged forty-four years, had enjoyed excellent health up to the period of the first attack of cardiac distress, which was sudden and unexpected. His habits were temperate but sedentary, and, from being confined to his desk during six days in the week, he could only take exercise on the Sundays, when he made walking excursions to the country. On one of these occasions he was attacked with dyspnœa, palpitation, and præcordial oppression. He was soon afterwards seen by Dr. Fleming, who found the pulse weak, small, and irregular, intermitting, while the impulse of the heart was strong and much extended. A loud bellows murmur was found attending the second sound of the heart.

The attacks of cardiac distress became frequent, and were produced by bodily, or even mental exertion. A fremitus was found to attend the murmur, which latter was very distinct in the interscapular region, and at one point indeed louder than in the front of the chest.

But the most important feature in this case was the frequent occurrence of marked cerebral symptoms, very similar to those observed in cases of fatty degeneration of the heart. These attacks generally came on at night, or during sleep, the symptoms being that the respiration would become suddenly stertorous, with some convulsion of the face, when the patient would awake, perfectly paralyzed at the left side. Jaundice also attended these attacks; and it was most remarkable that both the hemiplegia and jaundice would subside in a very short time. The full power of the muscles would return within a few hours after the attack, and on the following day scarcely a trace of jaundice could be seen.

It was found that these attacks were only to be treated by the use of stimulants. During one of them—owing to a different course having been adopted in the absence of Dr. Fleming—the patient was brought into the most extreme state of collapse. The stimulants had been withheld, and the head blistered; but, even under these circumstances, so decided was the effect of stimulants, that the patient, who in the morning was completely hemiplegic, was within six hours perfectly restored to the use of his limbs. This treatment was adopted in all the subsequent attacks, and consisted in the use of wine and brandy, together with the application of sinapisms to the region of the heart. The patient could not bear the slightest reduction, and showed a remarkable susceptibility to the action of opium.

The heart was found to be much enlarged, owing principally to a great increase in size of the left ventricle; all the cavities, and also the aorta, were filled with blood. The general form of the heart was remarkable; it was globular, the apex appeared wanting, and the left ventricle at its margin represented the segment of a circle. The right ventricle was very small, having not more than a third of the capacity of the left, the parietes of which were thickened, though not to any very great degree. The aortic valves were perfect. The mitral orifice was much enlarged. The circular cartilaginous ring was fully the diameter of a crown-piece; so that the valves, which were thickened, were quite incompetent to close it. The foramen ovale remained open, although by a very small orifice, which was oblique and valvular towards the left auricle.

In this case the cerebral symptoms were, doubtless, of the same nature as those which occur when the left ventricle is the seat of fatty degeneration; and future observations must determine how far the open and dilated condition of the mitral orifice may have tended to produce the effect in question, by diminishing the arterial supply at each systole of the heart. Indeed, if we exclude the stethoscopic signs, it may be said that, had the impulse of the heart in this case been feeble, all the symptoms of fatty heart would have been present in an extreme degree.

It has been thought that the presence or absence of paralysis would serve to distinguish the true cerebral apoplexy from that false or pseudo-apoplexy which occurs in cases of deficient supply. It is true that, in most cases of fatty hearts, the cerebral attacks have not been followed by paralysis; yet, in a few, paralysis has been observed; and in the case now given this condition attended every attack, subsiding, however, with great rapidity; none of the indications of chronic disease of the brain occurred in this case.

The appearance of jaundice with each of the attacks, which, like the hemiplegia, was ephemeral, is to be noted. Jaundice, as attendant on contraction of the mitral valve, has been described, but I do not know any instance of the repetition of a jaundiced state, such as was observed in this case.

In connexion, however, with this subject, the following observation has some importance: A lady, aged about forty, of a spare habit, complained of an itching of the skin, which was often so severe as to deprive her of sleep: soon afterwards her skin assumed a semi-jaundiced tint, and she sought for medical advice. The pulse was permanently rapid, though small, and yet there were no symptoms of fever. The action of the heart was excited and the arteries of the neck were observed to throb with force. Soon afterwards the thyroid gland became enlarged to about the size of a hen's egg.

The jaundice and itching of the skin continuing, the patient was put under treatment for an affection of the liver, but no impression was made on the symptoms. It was after this period she consulted me. But little change had occurred in the symptoms. The pulse was rapid and small; and though the swelling of the thyroid had declined to a great degree, more or less throbbing of the carotids continued; yet the pulse had none of the characters observed in permanent patency of the aortic valves. A loud bellows murmur attended the first sound, most distinct between the nipple and lower portion of the sternum. No tumour of the liver could be discovered, but the semi-jaundiced condition varying in amount from day to day, remained, notwithstanding a decided mercurial treatment. The alvine evacuations were always clay-coloured, but the urine remained of its natural appearance; a combination of circumstances which I never before witnessed, and which has now continued for several months.

The history of jaundice and of affections of the liver, in connexion with disease of the heart has yet to be written. That the jaundice in this case was consequent on organic disease of the heart there can be little doubt; and in this example, as well as in that by Dr. Fleming, it was present under circumstances of great peculiarity.

Before concluding these observations on enlargement of the mitral opening, we should note that, in Dr. Fleming's case, the left ventricle was found to be dilated, a condition very different from that observed in simple contraction of the orifice, so that inadequacy of the valves may be followed by dilatation of the ventricle, no matter whether the mitral or aortic orifice be the seat of the lesion. In the case of the mitral valves, with an actually enlarged opening, the ventricle and auricle may be held to form one bilocular cavity, both portions of which have a mutual re-action. The auricle having become distended, and probably hypertrophied, by regurgitation from the ventricle, sends an increased quantity of blood into that cavity, which latter has to expend its force not only in the direction of the aorta, but also in that of the auricle; thus it becomes not only dilated but hypertrophied; yet as the quantity of blood propelled into the aorta must be reduced in proportion to the size not only of the auricle, but also to that of its orifice, we have produced those effects which result from a weakened ventricle, even when no valvular lesion exists, as in fatty degeneration of the left ventricle.[1]

A comparison between the symptoms and anatomical results of the contracted and dilated conditions of the left auriculo-ventricular opening is still a desideratum. It may be suggested whether, in those cases in which a loud mitral murmur continues for many years without apparent injury to health, the condition of parts is at all events not a contraction of the orifice; it may be that it is dilated, or that its natural diameter is little, if at all, altered. This much is certain, that in such cases the indications, of pulmonary congestion, and of hypertrophy and dilatation, are for a long time absent, and the course which should be adopted is to preserve the general health; and while we take measures to avoid undue excitement of the heart, we must be especially careful not to depress its energy by an undue amount of antiphlogistic treatment. In fact, the principles laid down by Dr. Corrigan for the treatment of cases of permanently patent aortic valves are applicable in every respect to the condition which we have now specified.

Although in a large proportion of cases of disease of the mitral orifice there is regurgitation, even with a contracted opening, yet we must admit a class in which this regurgitation becomes an important condition, causing certain anatomical changes in the cavities, and producing manifest symptoms. In such cases the orifice remains of its natural dimensions, or becomes actually dilated. We do not know any physical signs by which these conditions can be distinguished from ordinary mitral disease, for the murmur in cases of this lesion is probably regurgitant. But in some examples of well-marked mitral murmur we find that it is not perceived in the interscapular region, while in others it is distinctly heard along the spine, sometimes, indeed, louder in this situation than in the front of the chest. In such cases the orifice is probably but little contracted, or, it may be, actually dilated. We cannot, however, take this interscapular murmur as diagnostic of free regurgitation, inasmuch as it may occur in a contracted state of the orifice. An example of this has already been given.[1]

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[1] Dilatation and hypertrophy of the left ventricle are noticed by Dr. Walshe as attendant on regurgitant disease of the mitral orifice. Among the causes of insufficiency of the valves, the enlargement of the orifice, without coeval growth of the valves, is considered by him to be of very rare occurrence. (Op. cit. p. 222.)

The loud systolic murmur heard along the dorsal region of the spine is, in most cases, indicative of very chronic disease. I once observed it to supervene in a case of endo-pericarditis, and to become permanent, although the ordinary signs of valvular disease had subsided. I have suggested that, in many of those cases where a mitral murmur continues for years without disturbance of the general health, the condition of the valve is not one of contraction. In such cases the interscapular murmur may often be found. Fremitus, too, appears more frequently associated with the murmur in these cases than in those of ordinary contraction; and when the dilatation is extreme, as in the instance of enlargement of all the orifices already given, and also in the example recorded by Dr. Fleming, it may become a prominent sign.

DISEASE OF THE AORTIC VALVES.

Comparing the diseases of the aortic with those of the auriculo-ventricular and pulmonary valves, we do not find that they have any special anatomical character. And in a mechanical point of view the effects of disease in this situation are the same as in the others. Thus, inadequacy of the valves is the most common result, existing with or without a contracted state of the orifice.

We have already pointed out three cases of the disease of the aortic opening in which, with great probability of accuracy, we may make a special diagnosis; these were as follows:—

1. Permanent patency of the valves, in which the diameter of the orifice may be increased or diminished, or remain in its natural condition.

2. An extreme amount of ossific growth surrounding the ori-

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[1] See page 204.

fice and stretching irregularly into the ventricle: here the valves are often destroyed.

3. Earthy or atheromatous deposit on the ventricular face of the valves, which latter, however, are still competent to close the orifice. This condition is often seen in connexion with fatty degeneration of the left ventricle.

It will be unnecessary to enter into detailed descriptions of other pathological conditions of the valves, such as their atrophy, producing a cribriform state, or those examples of dilatation of the orifice which result from enlargement either of the ventricle or of the aorta.

The most important, because the most frequent, of all these lesions, is that in which, from regurgitation of blood, such important consequences follow, and such characteristic signs are produced.[1]

At the commencement of this chapter it will be recollected that the diagnosis of this disease, founded on the observations of Dr. Corrigan, was given in outline. The following extracts from the original memoir of that accurate and distinguished observer being studied, we shall be in a position to take a general view of this disease of the heart.

After alluding to the obscurity of the symptoms, Dr. Corrigan observes, that what is deficient in the general symptoms is amply supplied by the certainty of the physical signs. He specifies,—

1st. Visible pulsation of the arteries of the head and superior extremities; 2nd. *Bruit de soufflet* in the ascending aorta, carotids, and subclavians; 3rd. The fremitus, or rushing thrill felt by the finger in the carotid and subclavian arteries. In conjunction with these he notes the character of the pulse, which is invariably full.

"When a patient affected with this disease," says Dr. Corrigan, "is stripped, the arterial trunks of the head, neck, and superior extremities immediately catch the eye by their singular pulsation. At each diastole the subclavian, carotid, temporal, brachial, and in some cases even the palmar arteries, are suddenly thrown from their bed, bounding up under the skin. The pulsations of these arteries may be observed in a healthy person through a considerable portion of their tract, and become still more marked after exercise or exertion; but in the disease now under consideration the degree to which the vessels are thrown out is excessive. Though a moment before unmarked, they are at each pulsation thrown out on the surface in the strongest relief. From its singular and striking appearance, the name of *visible pulsation* is given to this beating of the arteries. It is accompanied with *bruit de soufflet* in the ascending aorta, carotids, and subclavians; and in the carotids and subclavians, where they can be examined by the finger, there is felt *fremissement*, or the peculiar rushing thrill accompanying with *bruit de soufflet* each diastole of these vessels. These three signs are so intimately connected with the pathological causes of the disease, and arise so directly from the mechanical inadequacy of the valves, that they afford unerring indications of the nature of the disease. In order to understand their value, it is necessary to consider their connexion with the cause by which they are produced. The visible pulsation of the arteries of the neck, &c. may be first examined.

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[1] Edinburgh Medical and Surgical Journal, vol. xxxvii., pp. 227, 228.

"In the perfect state of the mechanism at the mouth of the aorta, the semilunar valves, immediately after each contraction of the ventricle, are thrown back across the mouth of the aorta by the pressure of the blood beyond them, and when adequate to their function of closing the mouth of this vessel, they retain in the aorta the blood sent in from the ventricle, thus keeping the aorta and larger vessels distended. These vessels consequently preserve nearly the same bulk during their systole and diastole. But when the semilunar valves, from any of the causes enumerated, become incapable of closing the mouth of the aorta, then, after each contraction of the ventricle, a portion of the blood just sent into the aorta, greater or less, according to the degree of the inadequacy of the valves, returns back into the ventricle. Hence the ascending aorta and arteries arising from it, pouring back a portion of their contained blood, become, after each contraction of the ventricle, flaccid or lessened in their diameter. While they are in this state, the ventricle again contracts and impels quickly into these vessels a quantity of blood, which suddenly and greatly dilates them. The *diastole* of these vessels is thus marked by so sudden and so great an increase of size as to present the visible pulsation which constitutes one of the signs of the disease.

"That this visible pulsation of the arteries is owing to the mechanical cause here assigned is made evident by several circumstances. It is most distinct in the arteries of the head and neck, which empty themselves most easily into the aorta, and of course into the ventricle. In the arteries of the lower extremities, of even larger size than those which present it about the head and neck, it is not seen to any comparative degree, and most generally not at all while the patient is standing or sitting. It is much more marked in the arteries of the head and neck in the erect than in the horizontal posture."

Since the publication of Dr. Corrigan's researches, the experience of many observers has tended rather to confirm every part of his diagnosis, than to add any new information. Yet there are some collateral points which are deserving of study.

This disease, which appears to be one of middle life rather than of youth or old age, and more frequently met with in the male than the female, is, either in its isolated form, or combined with an affection of the mitral valves, of common occurrence. It may be met with in young persons after an attack of rheumatic carditis, and it is probable, that in a large proportion of the cases which occur under the age of twenty-five years, the exciting cause has been an endocarditis. On the other hand, the examples occurring in men from thirty to fifty years of age seldom show a distinct inflammatory origin.

In many of these cases a general morbid state is to be observed, to which it is difficult to give an appropriate term. It is a condition approaching to that which favours the deposition of fatty, atheromatous, and probably tuberculous matter, a condition of deficient hæmatosis,—induced often by excesses or over-fatigue,—and attended by a weakened state of the nervous system. That a connexion exists between the atheromatous diathesis and that in which fatty and tuberculous matters are deposited must be admitted, even although the researches of Andral, Lobstein, and Gluge, had not tended to the same result.

We have alluded to the diminished vital energy in this disease. This is shown, not only, as Dr. Corrigan has remarked, in the want of proportion between the impulse of the heart and the amount of hypertrophy of the left ventricle,—as well as in the injury done by an antiphlogistic treatment,—but also in the character of the local inflammations of other organs than the heart, to which the patients are liable. I have generally found that such inflammations were of a low kind; that they resisted ordinary treatment; that when, for example, pneumonia set in, which is not uncommon, it had a spreading, somewhat erysipelatous character, resisting local treatment, and not benefited by tartar emetic or mercury, especially the first. It is a common error for practitioners, when called to a case of acute bronchitis or pneumonia supervening on this condition of the heart, to overlook this peculiarity of constitution, and they are too often surprised at the rapid sinking of the patient, who, but a few days before, appeared to be in a safe position.

The injurious effects of a too severe antiphlogistic treatment in these cases is to be attributed, not only to the weakening of the left ventricle, the hypertrophy of which seems a provision of nature, but also to the fact, that the entire organism being under the influence of a depraved chemico-vital condition, is unfit to bear reduction, or respond favourably to the action of remedies.

If we consider the physical signs of this disease, which embrace not only the evidences of regurgitation through a diseased orifice, but of those of dilatation and hypertrophy of the left ventricle, we find that the diagnostics given by Dr. Corrigan apply essentially to the disease when, as it were, it is at its maturity; having, on the one hand, passed its first stage, and, on the other, not yet arrived at the period of depression of the action of the heart. At both these periods, in fact, the completeness of the signs may be found wanting. Thus, in the first stage, we may have the throbbing pulsation of the innominata, and of the carotid and subclavian arteries, with a systolic murmur propagated into these vessels, yet without the second or regurgitant murmur. And again, in the latter periods of these cases, the throbbing and visible pulsation of the arteries cease, at least in the radial artery, and, to a great degree also in the carotids, while the double murmur under the sternum remains, though with diminished intensity. Cases of this disease occur in which for a long time, the radial pulse has been characteristic, and yet, for many days before death, there may be nothing remarkable in the pulse at the wrist.

But, in strong contrast with that condition, where from the progressive enfeebling of the heart, the arterial throbbing is found to subside, and, as it were, retreat towards the heart, we must place a category of cases in which the signs go on augmenting up to almost the last period of existence. In such cases there is not only a greatly dilated and hypertrophied left ventricle (*cor bovinum*,) but the vital contraction of the organ is unimpaired, or, it may be, augmented; so that, from many causes, including, possibly, a dilated state of the aortic orifice, the most violent pulsations of the arteries all over the body are produced, and the whole trunk pulsates like one vast aneurism.

We may divide the cases of permanent patency of the aortic valves into those in which the heart's action is either not excited, or even depressed; and those where the enlargement of the left ventricle is attended by augmented contractile power; and it will be found that there is more chance of prolongation of life in these cases than in those of the former class, notwithstanding the greater prominence of the symptoms. This may arise from the disease being in one case accidental, as when it proceeds from an endocarditis; while in the other it is but a sign of a generally morbid condition of the system, of a special and essential disease, which, even if the heart affection had not occurred, would assuredly, although by some other process, shorten the life of the patient.

In most cases, however, the fatal termination is preceded by a gradual failure of the powers of life; and Dr. Corrigan has shown that, as the contractile power of the left ventricle becomes less and less, death may take place from the want of arterial supply. The death is commonly gradual, but may be sudden. I have already remarked, that sudden death in cases of this kind appears to be less frequent than in disease of the mitral valves.[1]

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[1] A case of sudden death, occurring in a patient aged 15, who for five months had laboured under the effects of inadequate aortic valves, was brought forward by Dr. Corrigan at a meeting of the Pathological Society, in December, 1841. In this affection the form of the heart differs remarkably from that observed in dis-

A remarkable difference between this disease and the contraction of the mitral orifice is the want of that irregularity of pulse which so often attends the latter affection. In the disease of the aortic valves we often observe that the pulse, though full, throbbing, and collapsing, is regular. In certain cases an occasional intermission occurs, but the general character of pulse, as to rhythm, and even frequency, is but little altered from the state of health. It is under these circumstances that the diagnosis is most easily made, for the physical signs are much more obscure when, with inadequacy of the valves, we have irregular action of the heart; the arterial throbbing and the to-and-fro murmur then become much less evident; so that, at particular periods of the case, the diagnosis of the special lesion is difficult.

It is probable that, in some of these cases, a double valvular lesion exists, and that the mitral as well as the aortic orifice is engaged. Yet even with the double lesion, the pulse may remain singularly regular.

We have already alluded to the occurrence of two forms of this disease, in which the difference of symptoms depends less on the imperfect state of the aortic valves than on the condition of the left ventricle, especially as regards its vital contractility and power. In the first class of cases, as Dr. Corrigan has shown, the symptoms are often obscure, and the disease might escape observation, unless by stethoscope examination, and the existence of visible pulsation of the large arteries. In the second class, however, we have the symptoms much better marked, and yet the disease is often of longer duration. I have suggested that

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ease of the mitral valves, as noticed by Dr. Adams (Dublin Hospital Reports, vol. iv.) in which, owing to the fact that the apex is formed by the right ventricle, the heart presents a somewhat globular appearance. In the case before us, however, as might be expected, the apex or the organ is chiefly formed by the left ventricle—(See also Dr. Law's observations on the same point: The globular form of the heart, however, is not peculiar to enlargement of the right ventricle, for it may occur in cases of isolated dilatation of either cavity, with or without hypertrophy. Of this, the case communicated by Dr. Fleming is a good example. Here, it will be recollected, the heart was globular, owing to the enlargement of the left ventricle. It would be an interesting investigation to determine whether inadequacy of the mitral valves produces a different result as to the form of the left ventricle from that observed in the open state of the aortic orifice.

in these instances the lesion has been originally accidental, not resulting from a morbid constitutional state. Of such cases, the following is an example, for which I am indebted to Dr. C. Croker King.

CASE XXIV.—*Extensive disease of the Aortic Orifice, with inadequacy of the Valves; Vast hypertrophy and dilatation of the left Ventricle, probably secondary to an attack of Endo-pericarditis; Aggravated symptoms of Angina Pectoris, continuing to recur for upwards of ten years.*

A gentleman, aged 29, of delicate habit, was attacked with symptoms of pleurisy, and in all probability, of pericarditis, seven years before the time that he consulted Dr. King. When the patient first came under notice it was plain that a great hypertrophy of the heart had been established, as a strong and extended impulse could be distinctly seen; the pulse was sharp and sudden, and occasionally intermitted. From the second left rib to the ninth there was dulness on percussion, extending to the right of the sternum, and bounded on the left by a line drawn perpendicularly from the centre of the axilla. The whole area of dulness was about thirty-six square inches. On placing the ear to the side of the chest a sensation was communicated which was compared by Dr. King to the blow of a bladder filled with fluid, accompanied by a peculiar sound, similar to that produced by placing the finger on the tragus, so as to close the external meatus, and then withdrawing it suddenly; this was terminated by a muffled bellows murmur. The second sound was attended by impulse and murmur, but the latter was much sharper and shorter than the systolic murmur; this was heard most distinctly at a point higher than the usual position of the aortic valves.

This patient suffered from paroxysms of angina pectoris, with an amount and intensity of suffering probably unprecedented.

The paroxysms were preceded by general nervousness, and increased palpitation, gradually augmenting until the heart's action became tumultuous, accompanied by a sensation of aching down the arms and legs, with a feeling of lassitude and a desire to sit down, which, however, the patient dare not do, for fear of inducing an accession of the paroxyms. After a variable length of time, perhaps two or three hours, spent in fruitless endeavours to ward off the paroxyms, it fairly set in with a sense of constriction referred to the sternum, as if that bone and the spine were being forcibly approximated, and a sensation of the heart being torn from the thorax. As the paroxysm proceeded, the aching pains in the arms were replaced by a sensation as if red-hot wires extended along the course, especially of the ulnar nerves; the heart beat with the most extraordinary violence, causing the whole frame to vibrate; the carotids appeared impatient of the restraint of the integuments, and every superficial branch in the body could be traced; at each stroke of the heart, the whole person appeared to undergo a general dilatation, as if it were one great aneurism.

In order to obtain relief, he was accustomed to throw his head back, and to extend the spine, as is seen in opisthotonos; the arms were stretched first downwards, and then elevated above his head to the fullest extent, in order to give the great pectoral muscles a fixed point of action, in the hope of relieving the sense of thoracic constriction. The position of the patient, his dark, wild, staring eyes, and pallid face; the intensity of his agony, the perspiration, which at first stood in large drops, and then ran down his neck, altogether embodied a scene which baffles description, presenting a picture of suffering which could not be imagined or described.

When this great excitement had subsided, he felt perfectly tranquil: he appeared like one relieved from some desperate struggle, and was full of vivacity, wit, and humour. When a paroxysm was to occur at night, the patient awoke with a sensation resembling night-mare, and started up from bed; the slightest exertion, such as merely throwing the quilt about him, was sufficient to bring on an attack, and on this account he was accustomed for a long time to sleep in his clothes. On many nights, worn out by his efforts to ward off an attack, and having been overcome by sleep in the erect position, he would fall to the ground, and start up in a paroxysm of unusual severity. Latterly the paroxysms became more and more frequent; the mere act of eating induced them, so that times he was afraid to taste food, or if he fancied himself so situated that he could not assume at will a posture of relief, this feeling was itself sufficient to induce a paroxysm; in fact, the slightest moral cause was enough to produce it: for a length of time he was unable to see any friend; he usually came down stairs at five o'clock, and if any person took notice of him, or inquired how he was, he at once got an attack. He walked about the room, or leaned upon the mantelpiece during dinner, never sitting down to a meal.

He had always experienced the greatest relief from stimulants, so that without any real desire for them, he was in the habit, for many years, of drinking daily eighteen tumblers of punch—an attack of delirium tremens, however, determined him to abandon this custom, and to substitute opium; by great management and forbearance he restricted himself to one pint of laudanum in the week, provided it was made of the best opium. None of the salts of morphine, or even the black drop, except in very large doses, produced the effect desired. Other stimulants, for instance, Hoffman's anodyne, if combined with the salts of morphine, afforded relief. About every six months he suffered from partial suppression of urine, accompanied by pain across the region of the kidneys. Towards the close of the case, anasarca, confined exclusively to the lower extremities, set in; there had never been any puffiness of the face, but it is to be remembered that he did not lie down, as the recumbent position appeared to impede the heart's action; there never was dyspnœa nor cough. He was at length found dead in his bed, after having been seen about an hour previously in his usual position, sitting, or rather propped up, in bed, when he expressed his satisfaction at having passed a good night. The servant, on returning about an hour afterwards, found his master dead.

His death, it would appear, had been perfectly easy, as he was in the same position as when last seen during life.

During the entire progress of the case, which was of ten years' duration, there had never been the least evidence of congestion, local determination of blood, or interrupted circulation. No epistaxis, hæmoptysis, suffusion of eyes, headach, or frightful dreams, occurred; nor was there, as before remarked, the slightest cough or dyspnœa.

At the post mortem examination, thirty hours after death, on throwing up the sternum and cartilages of the ribs, an immense pericardium alone presented itself, which was found to be universally adherent to the surface of the heart, thus corroborating the opinion formed as to the origin of the disease. The base of the heart was situated in an unusually high position; the left ventricle was hypertrophied and dilated to an extraordinary degree; the weight of the heart, after the coagula were removed, being forty-four and a half ounces. The hypertrophy was confined to the left side; the right ventricle did not nearly reach the apex of the heart; in fact, not the apex alone, but almost all the lower part of the heart, was formed by the left ventricle; the sinuses of the aortic valves were almost filled by rugged calcareous deposits. The double *bruit*, alluded to at the commencement of the case, was evidently produced as follows:— The soft, prolonged, first *bruit*, by the passage of the blood over the cardiac surfaces of the valves, while the roughness of the second *bruit* was due to the regurgitation of the blood over the roughened arterial surfaces of those valves, the calcareous deposit having taken place at their aortic surface. The shortness of the second *bruit* might be accounted for by the rapidity of the heart's action, as the pulse generally averaged 120, so that the frequency of the ventricular systole prevented a long duration of the regurgitant murmur; the aortic orifice was perfectly free, though the valves were inefficient; the aorta itself appeared to be thinned and slightly dilated. The kidneys were enlarged, slightly indurated, and mottled, presenting a number of minute asperities.

The dissection did not reveal any further change to which the immediate cause of death might be attributed. There was no extravasation into the brain. Taking into consideration the undisturbed position of the body, as well as other circumstances, Dr. King inclines to the opinion that the death was caused by syncope.

Any medicine of a depressing nature, such as digitalis, was sure to aggravate his distress; saline purgatives had also a similar effect.

From the enormous size of the heart it might naturally be expected that the corresponding part of the chest would be prominent, but it was, on the contrary, flattened.

SIMULATION OF ANEURISM.

As might be expected, the disease is often mistaken for aneurism of the aorta, or innominata; an error not only injurious to medicine, but productive of the worst consequences to the patient. I have known this error to be more frequently made in cases where the disease was recent, and exhibiting well-marked impulse at the upper sternal region within a short space of time. On the other hand, Dr. Corrigan gives an example of very chronic disease in which the pulsations in the region of the innominata were so strong, that no doubt was ever expressed that the case was not one of aneurism. On dissection, it was found that the aorta was thinned and dilated, so as to cause imperfection in the closing of the valves, and the dilatation extended to the innominata, carotids, and subclavian arteries. This author well observes, that, "an acquaintance with the disease under consideration, and a knowledge of the fact that a violent throbbing at the root of the neck, or notch of the sternum, may arise from another cause than aneurism, will prevent the forming of a rash opinion on the cause of the violent throbbing. This throbbing may proceed from aneurism, or may arise from inadequacy of the aortic valves. When it proceeds from aneurism of the arch, or of the *arteria innominata*, it is confined to the vessel or the region of the vessel affected; the other trunks arising from the arch present only their natural, or even a diminished pulsation, and there is in the trunks arising from the arch neither *bruit de soufflet* nor *fremissement*. On the contrary, when the throbbing at the notch of the *sternum*, or in the region of the *arteria innominata*, is from inadequate aortic valves, all the larger trunks arising from the arch pulsate in an equal degree, or with trifling differences, arising merely from the relative sizes of the vessels, or their relation to the surface, and they are never at any time without *bruit de soufflet* and *fremissement*.

"Not only in relation to treatment, but in regard to the patient's mental anxiety, it is of importance to be aware, that inadequacy of the aortic valves may, by this violent pulsation at the root of the neck, closely simulate aneurism of the arch of the aorta, or the root of the *arteria innominata*. In aneurism of the aorta, life is not for a moment secure, and it may be necessary that even for a remote hope of cure the patient should totally abstain from all exertion. In permanent patency of the mouth of the aorta the fatal result is never sudden; and, under proper restriction, the patient is not only able to lead an active life for years, but is actually benefited by doing so."[1]

But aneurism and permanent patency of the valves occur in combination. When the diagnosis of aneurism comes before us we shall return to this subject, and here only remark, that the error of taking the disease of the aortic valves for aneurism arises not only from want of knowledge of the former disease, but from inaccurate notions as to the signs and history of aneurism itself. Thus, many believe that bellows murmur is always present in aneurism, and hence take it as a sign of the disease. And again, it is held that aneurism necessarily produces hypertrophy of the heart; and so this condition, so constantly present in permanent patency of the orifice, is held as an additional proof of the existence of aneurism. Yet the occurrence of bellows murmur in the artery, combined with the signs of hypertrophy of the left ventricle, which is the rule in permanent patency, is any thing but constant in aneurism.

I have known this disease to be mistaken for aneurism of the abdominal as well as the thoracic aorta. When we consider, that in confirmed cases of this disease, with an active left ventricle, all the arteries exhibit an increased pulsation, and recollect the law of the production of increased action of vessels in the vicinity of organs when in a state of irritation, we can understand how it might happen that in a person already labouring under increased action of the abdominal aorta, a local augmentation of that action would give rise to extraordinary pulsations, simulating aneurism of the abdominal aorta. In such a case, too, should there be an enlargement of the left lobe of the liver, we may have, for a time at least, a violent pulsating tumour in the epigastrium; yet it may happen that in a few days the symptoms may subside, and the patient, if his system has not been disturbed by reducing treatment, or his mind agitated by being told that he has so terrible a disease as abdominal aneurism, be restored to his ordinary condition.

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[1] Edinburgh Medical and Surgical Journal, Vol. xxxvii., pp. 236–237.

In such a case the attention of the practitioner must be directed to the following points:

1. The absence of the usual symptoms of abdominal aneurism.
2. The fact that the bellows murmur is not confined to the vessel supposed to be the seat of aneurism, but is heard in the thoracic aorta, and at the base of the heart.
3. The throbbing pulsation of the femoral arteries, which, as in the case of the carotids, may also present murmur.

Finally, he should suspect that the disease was not aneurism, from the existence of symptoms of constitutional irritation.

We might inquire whether, under circumstances similar to the preceding, an aneurism of the thoracic, as well as the abdominal aorta, might be simulated. On this point I have no observation to bring forward, and indeed there is less probability of sympathetic excitement of the artery in the thorax than in the abdomen, in which we see so many examples of excited action, even without inadequacy of valves. It is common in hysteria, and may be met with in various irritations of the digestive system, or as an attendant on menstruation or the earlier stages of pregnancy.

As bearing on the history of augmented local action of arteries, with previous inadequacy of the aortic valves, the following case is important. The patient was under the care of Dr. Graves and myself during the greater portion of his long-continued and extraordinary ailment.

CASE XXV.—*Long-existing signs of Inadequacy of the Aortic Valves; Persistence of symptoms simulating Rheumatic Fever; Local Arterial Excitement: Cessation of Pulsation in the left Radial Artery; Death.*

A boy aged ——, who had for many years presented signs of a permanently patent aortic opening, was attacked by the illness which terminated his life in the beginning of March, 1851. The period of commencement of the disease of the heart could not be accurately determined, but that a to-and-fro murmur had existed at the base of the heart for many is certain. His last illness commenced by symptoms resembling gastric irritation, of a remitting character, attended with irregular shivering fits, which continued to recur for a great length of time. The first indication of anything like rheumatic disease was the sudden supervention of pain in the calf of the leg. The paroxysms of shivering sometimes occurred within a few hours of one another, and were succeeded by high fever, during which the pulse at the wrist was singularly hard and thrilling; yet the action of the heart, although it was to a certain degree excited, was not proportionate. He complained much of the pulsation and noise in his head; and on one occasion he said that he felt as if his brain was acted on by a churn-dash. These symptoms were aggravated by the use of opium.

Soon after this period the disease assumed a character which it preserved with singular constancy up to the period of death. The patient was liable to attacks of shivering, followed by high fever and perspiration; almost every one of which was attended with a local irritation, simulating arthritis, and yet having this character, that the inflammatory action was more in the vicinity of the joint than in the articulation itself. The intervals between the rigors varied from eight to forty-eight hours, and no treatment had any effect in controlling the disease. This patient never presented any true form of arthritis. Thus, when the ankle appeared to be attacked, it was found that there were no signs of effusion into the joint, but the swelling, heat and soreness engaged the dorsum of the foot; so, also, when the knee was complained of, there was no tumefaction of the articulation, but a space of two or three square inches above the patella was the seat of disease. When the hand was engaged, it was along the metacarpal bones rather than in the joints, that swelling and tenderness were perceived. Finally, the local irritation consequent on each attack of shivering sometimes appeared in the most unusual situations,—the eyelid, the nose, and the insertions of the nails, were often the seats of this ephemeral irritation.

In the earlier periods of this singular case, the action of the heart was occasionally excited, but this liability disappeared, and the organ remained singularly tranquil, though still presenting the double bellows murmur propagated into the arteries. One of the most remarkable circumstances attending the case was the extraordinary excitement of the arterial pulse in the vicinity of the various local irritations. It is utterly impossible to convey in words any idea of the character of the pulsation, as observed in the anterior tibial artery and its branches. When the foot became engaged, we had then, with a tranquilly acting heart, and a feeble and compressible radial pulse, a pulsation so vehement and sharp that the impulse might be compared to the blow of a steel hammer on an anvil, conveying the idea that the whole foot was on the point of being burst and torn to pieces at every throb of the artery.

The disease having continued unmitigated for three months, it was observed that after one of the attacks in the left hand, the temperature of the arm was found to be much reduced, and the pulse at the wrist to have become very small and indistinct. Voluntary motion remained. A fortnight before this he had had a severe attack of pain in the left biceps. We soon found that no pulsation could be detected in the fore-arm, and that it was hardly perceptible in the upper portion of the brachial artery. In the course of about a fortnight, a feeble pulsation returned at the wrist; but there was no arrest of the fell disease which was consuming the patient. It continued to repeat itself, from day to day, with but little change, until at length the sweats became colliquative. Diarrhœa set in, and signs of congestive pneumonia closed the struggle, which continued for a period of nearly four months, resisting all treatment. The rigors occurred about every second or third day, until the last month of his disease, when the fever become more continued; and every rigor was followed by the peculiar local irritations, attended with the extraordinary local arterial throbbing—now in one part of the system, and now in another. There was no dissection.

Whatever may have been the nature of this disease, which resisted the use of bark, opium, mercury, iodine, colchicum, and stimulants, the case is eminently instructive as an example of local excitement of arteries to an extraordinary extent, occurring in connexion with ephemeral irritations, and in a case of permanently patent aortic opening of long standing.

There are few conditions more obscure in their nature than the local excitement of arteries, and few symptoms more singular than this local excitement, when it arises in a case of inadequate aortic valves.

That this disease was not arthritis is certain; and I cannot even offer a suggestion as to its nature, unless that we might suppose it to have been some form of erratic or metastatic arteritis.

With regard to the duration of the first stage of a disease which is to end in permanent patency of the aortic valves, there is a great variety observed. We meet with patients somewhat advanced in life, whose appearance indicates their liability to disease of the heart; they are generally of a full habit; they suffer from dyspepsia, and often exhibit a tendency to gout. Under the influence of temporary derangement of the stomach, these patients may complain of throbbing in the head, and of uneasy sensations, which draw attention to the state of the heart, when it is discovered that the pulse is hard, yet without the collapsing character observed in permanent patency of the aortic valves. The arterial pulsations are not visible, and it often happens that the symptoms may be removed, even for a long period, by treatment directed to the digestive system. Yet these patients present a permanent valvular murmur, which is systolic, but single, and propagated into the aorta; it is loudest at the base of the heart, and frequently absent to the left of the nipple, the second sound remaining clear. Such patients may continue in this state for a great length of time, and enjoy an excellent state of health, and are often able to take active exercise without distress of respiration. I have at present under my care a gentleman who has for upwards of two years laboured under this disease, yet who is able to enjoy the most active field-sports, and even walk up a long and steep hill without impediment to respiration. That such cases are of frequent occurrence I have no doubt; and the immunity from progressive disease of the heart seems to arise from this, that as the aortic valves remain competent to close the orifice, the patient escapes the effects of regurgitation.

The murmur in permanent patency of the aortic valves is generally double. It may, however, be single and systolic, or single and regurgitant. It is generally low and soft, and without musical tone. On the other hand, in cases of great and irregular ossifications at the mouth of the aorta, a musical murmur, propagated into even remote arteries, and sometimes so distinct as to be audible at a distance from the patient, may exist. But still, we may notice a loud musical murmur in connexion with the general signs of permanent patency. Professor Banks lately exhibited at the Pathological Society a specimen of diseased and inadequate aortic valves. Enormous vegetations, and masses of a soft atheromatous matter, filled the sinuses of the valves, and covered their ventricular surfaces. When water was poured into the aorta, it made its way into the ventricle, and there seemed a greater facility for regurgitation than for the passage of fluid in the direction of the aorta. A portion of this atheromatous deposit, more than an inch in length, with a narrow base, stretched freely upwards into the aorta, where it doubtless vibrated like the tongue of a Jew's harp. In this case a very loud musical murmur was transmitted along the aorta, and the arteries presented visible throbbing, as in the ordinary disease of the orifice. The patient died after a paroxysm of dyspnœa, the first which had occurred during the progress of the case.

DIAGNOSIS DERIVED FROM THE STATE OF THE CAVITIES.

Having now taken a general view of the diagnosis of valvular disease, as studied with reference to the practice of medicine, we may turn to the labours of Forget, one of the latest writers on diseases of the heart, and inquire how far he is justified in declaring that the law of retro-dilatation furnishes us with such fixed principles, as that its establishment should mark an advance in diagnosis. It is to be noticed, in the first place, that the doctrine of the liability to dilatation in the cavities of the heart, when, from obstruction, they are impeded in their efforts to empty themselves in the natural direction, is not new; indeed, the author observes that he does not claim it as such, but maintains that he has first established it on a firm foundation, and made it an important element in diagnosis.

The diagnosis of the seat of valvular disease at the left side of the heart, according to Forget, is easily attainable. He has shown the difficulty of distinguishing, by acoustic signs, between the affections of the right and left valves, a difficulty long before admitted by practical physicians. He maintains, also, that we cannot with safety determine the isolation of disease in the mitral or the aortic valves, if we confine ourselves to the study of the seat and character of sounds; so that, bearing in recollection the greater frequency of diseases of the left, as compared with those of the right valves, and assuming that a permanent bellows murmur, often rough and attended with fremitus, is the great indication of valvular disease, we are to conclude, that with such a murmur, the disease is in the aortic orifice when the left ventricle is dilated, and it may be, hypertrophied, and in the mitral valves, when the left ventricle is unaffected.

But in the case of mitral disease, the law of retro-dilatation is still in force. The left auricle becomes dilated, as indicated by dulness on percussion, and, subsequently, the right cavities of the heart. Again, the fulness of the præcordial region observed in active aneurism of the left ventricle will be wanting, the pulse will be small, and without the hardness and vibration which indicates increased power of the left ventricle. One diagnostic more is given, which cannot be admitted, namely, that the impulse of the heart is feeble. We know that when, from contraction of the mitral orifice, the right ventricle becomes enlarged, there is generally a strong impulse; and in connecting the dulness on percussion with feeble impulse, Forget has indicated two diagnostics, which seem incompatible.

But if we inquire whether the law of retro-dilatation has in reality such value, as that its establishment marks a step in advance in the science of diagnosis, a very doubtful answer must be returned. This dilatation *a tergo* is not constant, nor, when it occurs, can it be always recognised with certainty or facility. In how many cases of disease of the aortic valves are the signs of hypertrophy and dilatation of the left ventricle wanting? or, if we consider the contraction of the mitral orifice, by what means are we to demonstrate the dilatation of the left auricle? for there is a great difference between theoretical diagnostics and those justified by experience. A case is detailed by Forget, in which, in a patient aged 65, who laboured under chronic bronchitis, there were irregular pulsations with little impulse. A slightly rough bellows murmur attended the second sound, which was not propagated into the aorta. The diagnosis of disease of the mitral orifice was made. When the heart was displayed on dissection, the left ventricle was found to be greatly hypertrophied, upon which Forget immediately altered his diagnosis, and declared that aortic valve disease existed. The aortic valves were found ossified, shortened, and insufficient, while the mitral valves were healthy; and this case is quoted as a confirmation of the law. But we should have a larger knowledge before we designate as a law what is yet but the chance consent of a limited number of observations. Let us recur to the case by Dr. Fleming, and inquire how far the law of retro-dilatation would apply to it. Here was a case of valvular murmur, with a small, weak, and irregular pulse, and without signs of active enlargement of the left ventricle, but, on the contrary, with evidences of enfeebled power of the heart; and yet a great enlargement of the organ, owing almost entirely to the hypertrophy and dilatation of the left ventricle was found. In such a case, before the heart was opened, Forget would have made the diagnosis of lesion of the aortic valves, yet the disease was in the mitral opening, while the aortic valves were perfectly healthy and competent to close the orifice. It must be also borne in mind that retro-dilatation, is a condition consequent on the valvular disease, and that the period when it occurs to such a degree as to become available in diagnosis is infinitely varied in different cases: years may elapse with the existence of a valvular murmur before the cavity becomes dilated, and indeed, in some cases, death takes place by syncope, asphyxia, or rupture of the valves, without the signs of retro-dilatation having ever been manifested. We cannot say why in one case the cavities become hypertrophied and dilated, while in another an indisposition to this change appears to exist; and it is obvious that for the production of the change in question, something more than mere mechanical obstruction is necessary. There must be some vital alteration or organic change in the muscular structures, the presence of which favours the dilatation or hypertrophy, while its absence preserves the integrity of the cavities of the heart.

For the occurrence of a retro-dilatation must not be considered as merely a mechanical result of obstruction, nor that of retro-hypertrophy as a change necessary to overcome that obstruction. Great narrowing of the aortic opening may exist without hypertrophy or dilatation of the ventricle, a fact which is familiar to every pathological anatomist. I have seen more than one case in which, although the orifice was so narrowed as to make us wonder how the circulation was carried on, the left ventricle was unchanged. Professor Smith has met with several instances of this kind; and, on a late occasion, has found a contraction of the left ventricle (the concentric hypertrophy of authors) to coincide with extreme obstruction at the aortic orifice.

If, then, we reflect on these facts, and call to mind the many cases of valvular murmur continuing for years without the symptoms or signs of alteration of the cavities, and the circumstance that, even in the cases of retro-dilatation, the change is seconddary to the valvular lesion, we must hold that, in a large number of cases, we cannot avail ourselves of the signs of enlargement of the cavities in the diagnosis of valvular disease. Forget has not given sufficient weight to the influence of regurgitation in producing the dilatation and hypertrophy of the cavities. There is little doubt that it has an important effect in causing dilatation; and, so far as hypertrophy is concerned, its influence must also be considerable.

This much may be admitted, that, in cases of valvular murmur, the existence of signs of enlarged cavities is to be taken as corroborative evidence that the murmur indicates an organic disease of the valves. Considered with reference to the special diagnosis of disease of the aortic and mitral openings, all that Forget has established was announced long ago by Dr. Adams and Dr. Corrigan, the first of whom showed the value of the signs of enlargement of the right ventricle as a diagnostic of mitral disease; while the second established that hypertrophy and dilatation of the left ventricle was attendant on the permanent patency of the aortic valves.

There yet remain for consideration three forms of disease of the aortic valves. One of these, consisting of extreme ossification, with irregular growths stretching down into the ventricle, has been already noticed at the commencement of this chapter. Of this condition, the principal indications are the signs of an hypertrophied left ventricle, and the production of a musical murmur, which is systolic, propagated even into distant arterial branches, and often so loud as to be audible at a considerable distance from the patient.

The two remaining cases are distinguished by the existence, as a permanent condition, of a weakened left ventricle, often the result of fatty degeneration. In one case we have the regurgitant murmur of permanent patency, while, in the other, the murmur is single but systolic, and propagated into the aorta and its branches, the valves, though diseased, being competent to close the orifice. The leading characteristics of both these cases are the slow pulse and the repetition of the pseudo-apoplectic symptoms; but we shall defer their more full consideration until we speak of the fatty disease of the heart.

We may now state, in separate propositions, those conclusions, which have a practical importance with reference to valvular disease.

RECAPITULATION.

1. That cases of valvular affection may be divided into two classes, in one of which the disease has been produced by inflammation, while, in the other, it appears to rise independently of this condition.

2. That in the first class of cases, a period arrives in which, although the disease is progressive, there is no evidence of its being of an inflammatory nature.

3. That hence it is generally improper to persist in an antiphlogistic treatment of valvular disease beyond a certain period of time.

4. That the determination of the actual seat and nature of a valvular disease is of less importance than that of the vital and mechanical state of the heart.

5. That a permanently patent state of the orifices is the ordinary result of all valvular diseases. This condition may or may not be attended with contraction, or the orifices may be dilated.

6. That the period when inadequacy of the valves supervenes, varies greatly in different cases.

7. That hence, two series of phenomena may occur: in the first we have the signs of disorganization without inadequacy; in the second, those of inadequacy are added.

8. That the distinctness of valvular murmur cannot be taken as being proportionate to the amount of disease.

9. That a complete cessation of murmur may coincide with the advance of disease.

10. That the cessation of murmur, under these circumstances, has been only observed in connexion with contraction of the orifice; it has not been observed in cases of free regurgitation.

11. That absence of murmur does not necessarily imply absence of valvular disease, especially if there be symptoms of disease of the cavities.

12. That the number of cases in which we are warranted in making a special diagnosis of valvular disease is small.

13. That the number of pathological conditions competent to cause such changes in the valves as will produce murmur is very great.

14. That in the earlier periods of valvular disease, murmur may not occur, although the disease be progressive.

15. That even in chronic cases, the development of murmur may be sudden.

16. That the disorganizing process may advance with great rapidity, or with slowness, and that, in some cases, it appears to be really arrested.

17. That the irregular action of the heart is much more related to the state of the cavities than to that of the valves.

18. That we may observe the sudden development of the symptoms, as well as of the physical signs of chronic disease of the heart.

19. That three conditions of the heart, considered in its vital relations, may accompany or follow valvular disease:—

a. Increased force of the heart.

b. Diminished force, with rapidity and irregularity of action.

c. Diminished force, with remarkable slowness and comparative regularity of action.

20. That the law which regulates the production of the alte-

ration of the cavities, which follows on valvular obstruction, with or without inadequacy, is still undetermined.

21. That considering the rarity of organic change in the valves at the right side of the heart, and the difficulty or impossibility of their special diagnosis, we may, in a practical point of view, limit our considerations to the diseases of the mitral and aortic valves.

22. That in the diseased and permanently patent condition of the valves of the pulmonary artery, a double murmur at the base of the heart, not propagated into the aorta, and not attended with general arterial throbbing, has been observed.

23. That in most cases of organic disease of the valves at the right side of the heart there is either an open foramen ovale, or a deficient ventricular septum.

24. That the most frequent result of disease of the right auriculo-ventricular valves is but the exaggeration of their natural insufficiency.

25. That we cannot by the ordinary acoustic or tactile signs determine the existence of dilatation of the right auriculo-ventricular orifice.

26. That reflux pulsations in the veins of the neck, and occasionally in those of the upper extremities, indicate regurgitation into the right auricle.

27. That hence they may be taken as indicating the insufficiency of the valves, and may have, as their remote cause, morbid conditions of the pulmonary artery, the lung, or the left side of the heart.

28. That of these different lesions the most frequent is contraction of the mitral orifice.

29. That the venous pulse thus produced may be permanently present, or only developed during an attack of cardiac asthma.

30. That the pulsations in the jugular veins are synchronous and isochronous with the ventricular systole.

31. That we must not depend on any acoustic character of murmur, nor even on its exact seat, for the diagnosis of valvular disease. It is requisite to combine with these considerations those of the history and symptoms of the case, as well as those which

have reference to the state of the pulse, the force of the heart, and the condition of the lung and liver.

32. That all diagnostics depending solely on the tone, character, and seat of murmur, are more or less doubtful.

33. That although by acoustic signs we may often determine the insufficiency of a valve, yet there are no means by which, from the stethoscope alone, we can declare the cause of that insufficiency.

34. That the diagnostics between the contraction and dilatation of any of the orifices, founded on acoustic phenomena, are to be rejected.

35. That organic and anæmic murmurs may co-exist.

36. That there are no distinctive symptoms of disease of the mitral valves, when it is uncomplicated with alteration in the vital or mechanical state of the cavities.

37. That its principal physical indication is a murmur which is systolic, but not propagated into the arteries, and loudest towards the apex and to the left side. This may or may not be attended with fremitus.

38. That the most common result of contraction of the mitral opening is pulmonary congestion, with enlargement of the right cavities of the heart.

39. That under these circumstances, from the preponderance of the right ventricle, a globular form of the heart may be produced.

40. But the globular form of the heart may exist with a dilated mitral opening, attended with enlargement of the left ventricle, while the right remains unaffected.

41. That the combination of a contracted state of the mitral opening, with permanent patency of the aortic valves, is of frequent occurrence.

42. That under these circumstances, we may occasionally observe both the mitral and the aortic murmurs.

43. But that the absence of a mitral murmur, in a case of permanent patency of the aortic valves, does not necessarily imply that the auriculo-ventricular opening is free from disease.

44. That in cases of mitral contraction moveable coagula may

be formed in the left auricle, which may, by occlusion of the opening, become a cause of sudden death.

45. That with the progress of contraction the mitral murmur may gradually subside, and ultimately become extinct, so that with the increase of disease, we have decrease and cessation of murmur.

46. That this cessation of murmur may coincide with a permanently patent though contracted opening.

47. That inasmuch as most cases of mitral murmur are systolic, they are to be held as regurgitant. We cannot, by acoustic signs, distinguish between the direct constrictive and the regurgitant murmurs.

48. That the interscapular murmur may attend constriction or dilatation of the mitral opening, but appears more allied to the latter than to the former condition.

49. That the interscapular murmur may be consequent on a recent and acute disease of the heart.

50. That the existence of a pre-systolic murmur, which theoretically should imply that it attended the passage of blood from the auricle into the ventricle, does not justify the diagnosis of absence of regurgitation through the mitral orifice.

51. That the physical signs of the permanent patency of the mitral and that of the aortic orifice generally differ in this, that in the former case the murmur is single, in the latter double.

52. That in combination of disease of the aortic and mitral valves the whole of the mitral, and the first part of the aortic murmur, are the result of the ventricular contraction; the mitral being regurgitant, the aortic direct. But the second portion of the aortic murmur is regurgitant, and its corresponding phenomenon in the mitral opening, which, if it occurred, would be direct, is generally wanting.

53. That the pseudo-apoplectic symptoms, such as occur in fatty degeneration of the heart, may be also observed in cases of permanently patent and dilated mitral orifice.

54. That a murmur, loudest at the base of the heart and propagated into the arteries, indicates disease of the aortic valves.

55. That this murmur is single and systolic when the valves

are competent; but when they are inadequate it is generally double, but may be single and diastolic.

56. That the effect of regurgitation is to produce the signs indicated by Dr. Corrigan, namely, the visible arterial throbbing, the collapsing pulse, and the fremitus attending the pulsations of the arteries of the neck.

57. That in the progress of a case of inadequacy of the aortic valves three stages may be observed. In the first, the valves, though diseased, are still competent to close the orifice, and there is direct murmur propagated into the arteries, but without the visible throbbing of the vessel; in the second, we have the regurgitant murmur existing in the heart and arteries, together with the visible throbbing of the vessels, and increasing signs of enlargement of the left ventricle; while in the third we may observe, that while the to-and-fro murmur in the heart and the aorta remains, the pulse becomes less characteristic, and the visible throbbing subsides: this condition marks the gradual decline of the force of the heart and of the general strength, and indicates the approach of death.

58. That the duration of the first stage, or that preceding the permanent patency, varies in different cases.

59. That this disease may be induced by carditis, or arise independent of such a condition.

60. That cases of permanent patency of the aortic valves, originating in endocarditis, are more often met with in the young than in middle-aged persons.

61. That in many cases this disease seems to be secondary to a weakened state of the system at large.

62. That the local inflammations which may arise in cases of this affection have generally an asthenic character.

63. That in practice we may divide cases of this disease into two classes, the distinction being founded upon the state of activity or feebleness of the left ventricle.

64. That the disease may be mistaken for aneurism, not only of the thoracic, but of the abdominal aorta.

65. That in cases of permanently patent aortic orifice, the occurrence of local irritations, whether in the abdominal viscera or the extremities, may produce a localized and extraordinary

arterial throbbing, which disappears on the subsidence of its exciting cause.

66. That disease of the aortic valves, with or without inadequacy, may co-exist with fatty degeneration of the left ventricle, under which circumstances we observe a permanently slow pulse, with a murmur on the one hand, single and direct, and on the other, double, in consequence of regurgitation.

67. That it is occasionally met with in cases of feeble dilated hearts, when its diagnosis becomes more difficult from the smallness of the pulse and the rapid and irregular action of the heart.

68. That when evidences of dilatation of any of the cavities co-exist with valvular murmur, these evidences are calculated to strengthen the diagnosis of valvular disease.

69. That the signs of dilatation, with or without hypertrophy, will have still more value when the dilatation is manifest in that cavity in which the orifice of exit appears to be the seat of murmur.

70. But that the co-existence of a dilated left ventricle with valvular murmur may be observed in insufficiency and dilatation of the mitral opening, with healthy aortic valves, a pathological fact opposed to the law of retro-dilatation.

APPENDIX TO THE PRECEDING CHAPTER.

Since the introductory matter at the commencement of this chapter was printed, I have obtained the last edition of the Treatise by Skoda on Auscultation and Percussion; and as the views of this observer, as to the sounds of the heart, are worthy of careful consideration, and to a certain extent agree with those which I have put forward, no apology is necessary for the introduction of the following extract:[1]—

"*The two Ventricles, the Aorta and the Pulmonary Artery severally produce both the first and second sounds perceptible in the region of the Heart.*

"I believe that vivisections are not sufficient to solve the question of the origin of the sounds audible in the region of the heart, and that, to accomplish this, observations on persons in health as well as in disease, and careful comparisons of the phenomena observed during life, with the results of post-mortem examinations are indispensable.

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[1] Abhandlung über Perkussion und Auskultation, von Dr. Joseph Skoda, vierte Auflage. Wien 1850.

"An observer, whose ear is practised in auscultation, will, if he has the opportunity of examining many healthy and diseased individuals, find the truth of the following statements:—The sounds which depend on the motions of the heart, are not equally distinct and strong in different perfectly healthy individuals; in one they will be scarcely perceptible and not accurately defined; in another they will be, on the contrary, very clear, even in some measure ringing; in one case they can scarcely be heard in the cardiac region itself, while in another they are plainly audible over almost the entire anterior surface of the thorax, and even extend to the back: in many persons we hear these sounds particularly plainly over the part of the thorax against which the heart beats; while in others, the same region gives only indistinct tones, which, on the contrary, are much more plainly perceptible over the pulmonary artery and aorta.

"When we compare the sounds in the part of the thorax, against which the heart beats, with those heard above the base of the heart, in the situations under which the pulmonary artery and aorta lie, we shall often observe, that in the cardiac region the first sound, that is, the sound synchronous with the impulse, is longer than the second; but that, above the base of the heart, the accent falls on the second sound.

"If we compare the sounds in that part of the thorax, against which the apex of the heart strikes, and which corresponds to the situation of the left ventricle, with the sounds audible, at the same height, to the right of this point and beneath the sternum, that is, over the right ventricle, we sometimes observe that the sounds differ in the two situations, both in strength and clearness. In some cases I have also met differences in the pitch of the sound.

"Lastly, if we auscultate above the base of the heart, a little above the middle of the sternum, at the right edge of this bone, under which part the aorta runs, we will sometimes find the sounds to differ in strength and clearness, and in very rare cases, the pitch also, from those which we hear on applying the stethoscope at the same height, but about an inch to the left of the sternum.

"The modifications of the sounds in the parts I have pointed out, which are frequently perceptible in perfectly healthy individuals, are much more evident when we examine those who suffer from various morbid conditions of the heart. We should, therefore, first look for these differences in persons labouring under affections of the heart; and when we have once become familiar with them, we will also perceive the same in healthy individuals, in whom they are much less striking.

"If we have the opportunity of examining many patients in whom the heart is morbidly affected, we will meet cases in which neither first nor second sound is to be heard in the part of the thorax against which the apex of the heart strikes, corresponding to the left ventricle, in which cases we rather perceive in this situation a single or double bellows murmur, sawing, rasping, &c., while to the right of this point, corresponding to the right ventricle, and above the base of the heart, over the aorta and pulmonary artery, both sounds are plainly heard. In general, the sounds in the three situations are not similar in strength and clearness. In other cases, on the contrary, we have in the left ventricle, in the aorta, and pulmonary artery, both sounds frequently also differing from one another; while over the right ventricle nothing but a murmur is heard, which is synchronous with the systole of the ventricles.

Cases are yet more frequently met with in which no [normal] sound, but a single or double murmur, is perceived in the space corresponding to the course of the aorta; while both sounds are distinctly audible over the right and left ventricle, and over the pulmonary artery. It will also happen that we shall hear a single or double murmur over the left ventricle, and over the aorta, while we find both [normal] sounds persistent over the right ventricle and pulmonary artery; or we may hear murmurs over the left and right ventricle, or over the right ventricle and the aorta, or over the right and left ventricles and the aorta, and in the situations where no murmurs exist, we may find in some cases the normal sounds to be distinct, while in others they are indistinct or wholly absent.

"If these observations made upon innumerable occasions, and confirmed by others associated with me, be correct,—it appears to follow, with tolerable certainty, that both ventricles, the pulmonary artery and aorta, are capable, each separately, of producing both the first and second sound perceptible in the region of the heart.

"*The modifications of the sounds are frequently connected with variations in the state of the valves of the heart, and we must, therefore, in explaining the sounds, take into consideration the action of the valves during the motions of the heart.*

"If we compare a number of observations on living subjects with the results of post-mortem examinations, we cannot avoid the inference that the modifications of the sounds and murmurs are, in most cases at least, connected with the varying condition of the valves of the heart; for we generally find in a patient in whom we have observed murmurs instead of [the normal] sounds, abnormal conditions of the valves; excrescences, thickening, diminution, narrowing of the openings, &c. Yet it cannot be denied, that we sometimes find the valves in the dead body not exactly in the normal state, although during life there was no modification in the sounds, or only such as might possibly co-exist with a perfectly normal condition of the valves. Well-marked alterations in the sounds are not necessarily produced by every abnormal state of the valves; such changes may occur only in certain abnormal conditions of the valves, or other circumstances may co-operate with these conditions to produce the change in the sounds.

"It is by endeavouring to form a clear idea of what takes place during the motions of the heart, in the valves, as well in their normal as in their abnormal state, that we shall be able to distinguish the conditions which may be considered as possibly giving rise to the cardiac sounds, and as determining the modifications of these sounds and their change into murmurs. Through such a review of these conditions we shall obtain a guide for our observations, by means of which, or even by direct experiments, we may be able to separate what is real from what is merely possible.

"*Action of the bicuspid and tricuspid valves in the motions of the heart.*

"Laennec maintained that the columnæ carneæ are so connected with the valves that by their contraction they necessarily open them. He was, consequently, also of opinion that the columnæ carneæ do not contract simultaneously with the rest of the substance of the ventricles: that, on the contrary, their contraction ensues during the diastole of those cavities, so as to permit the flow of blood into them. Bouillaud, on the other hand, considers it quite manifest that the valve is closed by the contraction of the columnæ.

"We may draw the columnæ carneæ, and with them the chordæ tendineæ which spring from them, as strongly as we will in the direction they follow in the heart, without closing the valve, and the opening does not become smaller when the columnæ are placed more upon the stretch than when they are gently drawn. The shortening of the columnæ during their contraction will, therefore, not effect the closing of the valve. We also do not observe that in the relaxed state of the columnæ the blood is impeded in flowing from the auricles into the ventricles, and, consequently, their function is neither what Laennec thought, nor what Bouillaud supposed. Since the contraction of the columnæ does not determine the closing of the valve, no alternative remains but that the stream of blood itself, by pressing against the valve, effects its closing. The use of the chordæ tendineæ, which pass from the columnæ to the valves, is evidently to prevent the inversion of the latter, for, were the free borders of the bicuspid and tricuspid valves not firmly held by the attachment of the tendinous structures, the valves, during the systole of the ventricles, would be driven by the stream of blood partly into the auricles, partly towards the orifices of the arteries, and the closing of the valves could not take place.

"The chordæ tendineæ are distributed on the valves in a manner which is of the highest importance to the function of these valves, so much so, that without such an arrangement the bicuspid and tricuspid valves could not prevent the regurgitation of the blood from the ventricles into the auricles during the ventricular systole.

"From each columna carnea several stronger cords run to and are inserted into the middle of the ventricular surface of the valve, or some of them run to the base of the valve, and are inserted on the junction of the valve with the wall of the ventricle. From these stronger cords,—at about their middle,—and also from the columnæ, arise weaker ones, which are inserted somewhat nearer the free border of the valve. These latter serve as points of attachment to still more delicate ones, which are inserted nearer the free edge of the valve, and even on it. No chordæ tendineæ are attached to the auricular surface of the valve.

"If we draw the columnæ in the direction they follow in the heart, we will see that this puts only the stronger cords, which spring from the columnæ themselves, upon the stretch; the weaker, which do not take their origin from the columnæ, and are inserted near the free border of the valve, or on it, remain flaccid under the strongest traction. Consequently, we can never extend the free border of the valve by so drawing the columnæ carneæ; this is extended merely from its point of attachment to the point where the chordæ tendineæ springing from the columnæ are inserted. The entire remaining part of the valve, from the free border to its middle portion, remains flaccid.

"When we press back any point of this flaccid portion in the direction of the auricle, so that the cords which are attached to the part become extended, we see on it a number of pouches; and if we examine the entire valve in this manner, we shall be convinced that the ventricular surfaces of the bicuspid and tricuspid valves are not even, but exhibit pouches which begin immediately at the free edge of the valves, extend to the middle of their surfaces, or even further, and are manifestly formed in consequence of the peculiar mode of insertion of the tendinous cords.

"If we blow against the flaccid portion of the valve, towards the auricle, it will become inflated like a sail, and we may in this manner at once demonstrate the pouches in the entire circumference of the free edge of the valve. The same occurs when we pour water against the valve.

"When the blood endeavours, during the ventricular systole, to regurgitate towards the auricle, it must necessarily catch in the little semilunar pouches of the bicuspid and tricuspid valves, and swell the flaccid portion of the valve opposite the auricle to as great an extent as the chordæ tendineæ which are inserted into it will permit. By these distentions the passage to the auricle is closed against the blood, if the valve be held by the cords in such a direction that no opening shall remain after its distention. Hence the situation of the insertions of the cords on the walls of the ventricles, and their length, are not matters of indifference.

"The capacity of the ventricles is very different at the commencement of the systole from what it is at its termination, and the insertion of the columnæ carneæ during the progress of the systole are drawn nearer and nearer to the attachment of the bicuspid and tricuspid valves. In order that the length of the chordæ tendineæ should be adapted to close the valve, it is evident that those cords whose function it is to hold the latter in a proper direction must arise from such an arrangement as the columnæ carneæ.

"Thus, did they spring directly from the walls of the heart, they must, if their length were exactly right at the commencement of the ventricular systole, during its progress become too long, and, on the other hand, if they were only so long as to hold the valve in the proper direction at the end of the systole, they would obstruct the diastole. Since a change in the length of the chordæ tendineæ is impossible, they must necessarily be connected with muscles, and the use of the columnæ carneæ is evidently to keep the valve in the proper direction by their alternate contraction and extension. Thus during the progress of the systole the columnæ become shortened in proportion as their points of insertion approach the attachments of the bicuspid and tricuspid valves, an action which, were it not for the pressure of the blood, would maintain the chordæ tendineæ in precisely the same degree of tension they had at the commencement of the systole; and this tension would also continue unaltered during the diastole, in consequence of the columnæ becoming lengthened in proportion to the separation of the walls of the heart from one another.

"The correctness of the view here explained of the function of the columnæ carneæ appears to me to be corroborated by the fact, that the portion of the tricuspid valve situated on the septum receives its chordæ tendineæ only from very short columns, or directly from the wall of the heart. The points of insertion of these cords, in fact, approach the attachments of their portion of the valve but little or not at all during the systole, and, of course, are as little removed from them during the diastole. In this case a tendinous cord is sufficient to retain the valve, since no change in its length is necessary.[1]

"From what has been stated, the motions of the bicuspid and tricuspid valves would appear to be as follows:—During the contraction of the ventricles the valves are, by the shortening of the columnæ, prevented from being drawn out of these cavities and from approaching the mouths of the arteries. The columnæ and the chordæ tendineæ arising from them are at the same time drawn towards one another, and the surface of the valve to which the cords are attached becomes folded, and the opening of the valve is diminished.

"The remaining opening is closed by the portion of the valve which is not acted on by the shortening of the columnæ. This closing is effected by this part of the valve becoming filled, like a sail, with the blood which presses against it. The several points of the free edge of the valve come reciprocally into contact, and partly by the support they yield to one another, but principally by means of the chordæ tendineæ, the turning over of the free edge is prevented. As the delicate cords running to the free edge arise from the stronger chordæ tendineæ springing from the columnæ carneæ, all the latter stronger cords are brought into a curve by the action of the pressure of the blood against the inflated portion of the valve, which action is communicated by the fine cords attached to them.

"During the diastole of the ventricles the columnæ carneæ become lengthened and separated. The blood flowing from the auricle would press the valve against the walls of the heart, and partly towards the mouth of the artery, were it not retained by the chordæ tendineæ in its proper position. The chordæ tendineæ arising from the columnæ carneæ are, therefore, not relaxed during the diastole of the ventricles; for were they so, the valve might, at the beginning of the systole, not be in the direction required for instantaneous closing; a greater portion of the blood would always regurgitate from the ventricle into the auricle, and the valve should be drawn into its proper position, often against the stream of blood, by the contraction of the columnæ carneæ.

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[1] "The use of the columnæ carneæ, as here laid down, has already been described by Professor Weber, in Hildebrandt's Anatomie."

"In order that the bicuspid and tricuspid valves may perfectly discharge their functions, it is necessary that their free edge shall present the pouches I have described, and that the chordæ tendineæ and the columnæ carneæ shall have a length corresponding to the capacity of the ventricles. If the conformation of the valve be abnormal, it is either not in a condition to prevent the reflux of the blood from the ventricle into the auricle during the ventricular systole, that is to say, the valve is defective; or it opposes the passage of the blood from the auricle into the ventricle during the ventricular diastole.

"The former condition takes place in thickening and shortening of the free edge of the valve, or in adhesion of the latter to the chordæ tendineæ arising from the centre of the surface of the valve, by which the pouches are destroyed; in shortening or lengthening, or rupture of the chordæ tendineæ, in the formation of excrescences, the deposition of coagula on the edge of the valve and in adhesion of the surface of the valve to the wall of the ventricle; the latter condition, on the other hand, is produced by considerable excrescences, coagula of blood, or calcareous concretions, or the auricular surface of the valve, or by the adhesion of the chordæ tendineæ to one another and to the free edge of the valve, preventing the due action of the latter.

"*Action of the Semilunar Valves.*

"The semilunar valves of the aorta and pulmonary artery are, as is well known, pressed by the blood which is impelled during the ventricular systole into the artery, against the wall of this vessel, but during the diastole they are again distended by the blood, which is driven by the elasticity of the arteries forwards and backwards and towards the ventricles.

"By excrescences, calcareous concretions, &c., developed on the aortic valves, or by the adhesion of the three valves to one another, the latter are sometimes rendered immovable and incapable of being pressed against the wall of the artery, and they will thus obstruct the entrance of the blood into this vessel. If the free border of these valves be shortened or turned over, or be the seat of excrescences, if the valves be partially torn from their attachments or be perforated, they will not be in a condition to prevent the reflux of the blood, and the blood will, during the ventricular diastole, flow back from the aorta into the left ventricle.

"Whether the aortic valves have closed during life is very easily demonstrated in the dead body. If water be poured into an aorta, the valves of which are in their normal state, the fluid will not reach the left ventricle, but, being retained by the closed valves, will remain in the aorta, while if the valves be imperfect, it will flow into the ventricle.

"We possess no such test as to the state of the bicuspid and tricuspid valves. If we open the left ventricle at the apex, and, having tried the aorta, pour water through the opening, the passage of the fluid into the auricle will sometimes be prevented by the bicuspid valve. However, a repetition of the experiment will convince us that we have obtained no information as to the state of the valve. If we fill a ventricle with water, close its arterial opening, and then compress the ventricle, the bicuspid or tricuspid valve certainly becomes distended, but does not, even when its state is quite normal, completely prevent the reflux of the water. The reason of this is manifestly that the contraction of the columnæ carneæ and the multilateral diminution of the cavities of the heart cannot be imitated. We can only judge in the dead body whether the bicuspid or tricuspid valves have closed during life from the conformation of the valves, of the chordæ tendineæ, and of the columnæ carneæ, and from the changes which defect of those valves generally produces in the auricles.

"*Explanation of the Sounds in the Ventricles.*

"A comparison of observations on the living with the results of post-mortem examinations shows that a distinct first sound is rarely heard over the left ventricle when the bicuspid valve is not in a condition to prevent the regurgitation of the blood into the left auricle during the ventricular systole, i. e., when the bicuspid valve is defective. In such a case we generally hear a murmur synchronous with the systole, in the portion of the thorax against which the apex of the heart beats, while in all other parts of the cardiac region the first sound is plainly audible. The same is true of the right ventricle when the tricuspid valve has become defective. We then hear no distinct first sound over the right ventricle, although it is perceptible in the left ventricle, the aorta, and pulmonary artery, and in its stead we generally find a murmur to exist.

"The first sound in the ventricles, accordingly, generally arises from the sudden interruption of the stream of blood towards the auricle, in consequence of the dilatation of the bicuspid and tricuspid valves; also from the striking of the blood against these valves. Every impulse, as is well known, creates a sound, which is duller in proportion to the softness of the striking or of the stricken body. The tension suddenly effected in the valve by the pressure of the blood undoubtedly contributes to the production of the first sound; for fibres and membranes, when suddenly stretched, give rise to a sound,—not only in the air, as Gendrin and others believe,—but also under water. The fact that the first sound is often clear and clapping, and sometimes even ringing, seems especially to indicate that the stretching of the valves contributes to its production.

"It is manifest, however, that the first sound may sometimes also arise from the striking of the heart against the thorax. If in the dead body we strike the inner surface of the thorax with the finger, or with the apex of the heart somewhat firmly compressed, a clinking, or a sound differing but little from the ordinary first sound, will be heard through a stethoscope externally applied. If a part of the wall of the heart be, during the ventricular diastole, somewhat removed from the wall of the thorax, but during the systole strike it again, or even if the heart during the systole strike another part of the thorax than that against which it lies during the diastole, a clinking must likewise be produced, or a sound arise quite similar to the ordinary first sound of the heart; for the substance of the heart becomes hard during the ventricular systole. If the heart strike against the same portion of the thoracic wall on which it lies during the diastole, its impulse can produce either no sound or only a very dull one.

"The muscular rustling of the heart never occurs as a clapping sound, but merely as a dull protracted one, which I could never, in accordance with the phraseology I have adopted, designate a 'sound' (ton,) but must allude to as a noise approaching to the 'murmur.' This might be expected, for no muscle ever gives a defined, clapping, or ringing tone. I am not yet in a position to state, from observations on living subjects, whether the contraction of the substance of the heart is really attended by such a sound. The cases attended with violent impulse of the heart, and, consequently, with strong contraction of its substance, in which no first sound is audible, are not rare.

"The causes of the first sound now enumerated are not sufficient for all cases; all experiments particularly, hitherto made with a view to explain the modifications of the first sound, have proved imperfect.

"Greater difficulties attend the explanation of the second sound in the ventricles than of the first. It cannot be maintained that in the normal condition of the heart the second sound is always produced in the ventricles, for it is often probable, and not unfrequently certain, that the second sound heard over the heart arises in the arteries, and can, on account of its intensity, be heard at some distance. But there are certainly cases in which we are compelled to admit that the origin of the second sound is to be found in the region of the ventricle. Such are those cases in which the second sound is nearly absent or feebly perceptible over the base of the heart, while at the apex it is loud and clear. It cannot be conceived that such a second sound in the region of the apex is caused by the striking of the heart against the wall of the thorax, for this does not take place during the ventricular diastole.

"Perhaps the striking of the blood against the walls of the ventricle during the ventricular diastole may sometimes produce the second sound. In the left ventricle this impulse in a defective state of the aortic valves, and in a defective state of the bicuspid valve, is undoubtedly strong. Yet I have only in a single case, where the aortic valves were defective, found the second sound stronger at the apex than in any other situation; in this case it was certainly uncommonly strong and ringing. In a defective state of the bicuspid valve an increased second sound at the apex occurs more frequently.

"In constriction of the left ostium venosum we sometimes hear, instead of a protracted murmur with the diastole, two dull sounds over the left ventricle. This phenomenon Gendrin uses as the foundation of his explanation of the second sound of the heart, deriving the double second sound from the non-contemporaneous filling of the two ventricles. To me it seems more likely that the two sounds are part of a murmur which arises at the constricted part; that is to say, the murmur caused by the constriction is divided, when the action of the heart is feeble, frequently into two, but sometimes into three sounds. Further, in many cases, the murmur cannot be distinctly heard at one point, while around it two or three sounds, as it were the stronger periods of the murmur, are heard.

"*Explanation of the Sounds in the Arteries.*"

"In every large artery we can, in rare cases, hear a sound contemporaneous with the pulsation, which exactly resembles the sound of the heart. I do not think it can occur to any one to explain sounds heard in the crural or brachial artery by transmission from the heart; nor must we regard the sounds in the carotid and subclavian otherwise than as produced by these arteries, when there is either no sound perceptible in the cardiac region, or a weaker one than that heard in the neck. The latter phenomenon, especially, is frequently to be observed, but has generally been ascribed to a peculiar power of conducting sound, or has been left entirely unexplained. That the sound will be variously transmitted, according to the different condition of the thoracic viscera, is indubitable. But we will find cases enough in which the strength of the sounds above or below the clavicle, with weakness of the same sounds in the cardiac region, cannot be explained by the power of conducting sound, because the lungs are in a perfectly healthy condition. Bouillaud also ascribes a sound to the arteries, which he, however, does not state to be similar to a cardiac sound, but compares with the noise produced by the fingers when we give ourselves a rap on the nose. Certainly, the arteries distant from the heart give incomparably more frequently a merely muté sound, such as Bouillaud describes; the nearer ones, on the contrary, the carotid, subclavian, aorta, and pulmonary artery, give in general a sound as loud as those audible in the cardiac region; and, on the other hand, the sounds audible in the cardiac region are likewise sometimes mute.

"The sound audible in the arteries, synchronously with the pulsations, may be explained by the suddenly increased tension of the arterial coats. The second sound is audible in the aorta and pulmonary artery, and generally also in the carotid and subclavian. In the other arteries we rarely hear any sound coincident with their systole.

"The second sound in the aorta and pulmonary artery evidently arises from the shock of the column of blood contained in the arteries against the semilunar valves after the ventricular systole. The blood impelled by the systole into the elastic arteries is compressed by them, and so soon as the impulse from the heart has ceased, is necessarily driven rapidly back towards that organ.

"The current of the blood towards the heart is suddenly arrested by the semilunar valves. The shock which these suffer is communicated to the walls of the arteries, and not only is a sound produced thereby in the aorta and pulmonary artery, but this sound is also frequently heard in the carotid and subclavian, and, indeed, even when the aorta has lost the condition necessary to the production of a sound. This explanation of the second sound in the pulmonary artery and aorta is placed beyond doubt by observations on healthy and diseased subjects, and this sound appears to arise in no other way.

If the semilunar valves of the aorta have become defective, we hear no second sound over the aorta, but instead of it a murmur; the second sound continues, on the contrary, plainly audible over the pulmonary artery. If the coats of the pulmonary artery are abnormally distended, which must always be the case when the circulation in the lungs is overloaded, the second sound will be heard very much increased in strength over the pulmonary artery, while over the aorta it may be weak, or inaudible, or replaced by a murmur. The pulmonary artery, being strongly distended, presses with greater force upon the blood contained in it, and the shock of the column of blood against the semilunar valves is consequently more violent."

I have given the observations of Skoda in full, wishing to avoid the risk of misinterpretation had I given but an abstract of them. It will be seen that, in the general doctrine—that many causes concur in producing the sounds of the heart—his views and mine coincide. And although it is not yet proved that the ventricles, aorta, and pulmonary artery, are each capable of producing two sounds, yet there are grounds for such an opinion besides those which Skoda has mentioned. I have long thought that the double sounds in aneurism were difficult of explanation, unless on the supposition that a single cavity might produce a double sound; and we occasionally hear a perfect double sound in the carotids, which appears to belong to them specially. To this subject we shall return when we examine the diagnosis of aneurism.

With reference to the improbability of the sounds in the brachial or rather distant arteries being conveyed from the heart, the context appears to show that it is the double sound of the heart rather than any murmur, to which Skoda alludes. I have already shown, however, that a musical murmur, proceeding from disease of the aortic orifice, may be transmitted into the most distant vessels; and it is difficult to deny that if a murmur, originating in the very region of the valves, may be thus transmitted, that a sound (the second sound of the heart) might not occasionally be heard even in vessels more remote than the carotid or subclavian arteries.

I have not, in enumerating the possible causes of sounds in the heart, spoken of murmur produced by muscular contraction itself. Yet there are good reasons for believing that such may occasionally be produced. We often observe a peculiar sound in the heart, which is probably a murmur produced by the contraction of muscular fibre under particular circumstances. We find it during the period of recovery of the heart in cases of typhoid softening, especially in those instances where the first sound has at one time been extinct, when it gives a peculiar prolongation of the first sound, which has some resemblance to valvular murmur. We must, however, conclude, that it is a muscular murmur, not only from its acoustic character, but from its speedy subsidence as the heart's impulse is re-established, and the extreme rarity of valvular murmurs in typhus fever.

Skoda believes that the sudden contraction of the cavities has no part in producing the sounds of the heart. Where so many causes seem to concur in producing the first, if not both the sounds, it is difficult to prove that the ventricular contraction produces any part of the defined and suddenly produced systolic sound. Yet we cannot agree with him when he declares that muscular contraction never gives rise to a clear and defined sound. I have long been in the habit of exhibiting a simple mode of producing sounds in the voluntary muscles, very similar to those of the heart. If we insert a needle into a thick mass of muscle, such as the calf of the leg, and, having introduced another into any portion of the thigh, connect the two by bringing them into the current of a small galvanic battery, we find that the gastrocnemii muscles are thrown into clonic spasms, which continue for many seconds after the current has been interrupted. If during this period we apply the stethoscope, we hear not only the continuous though confused muscular sounds, but often well-defined sounds, which have characters singularly resembling those of the heart. If, then, under excitement, a solid muscle is capable of giving defined and sudden sounds, there seems no reason why similar results should not arise from the contraction of a hollow muscle, such as the ventricle.

But further: we find that in the cases already described, of disappearance of a valvular murmur consequent on the advance of mitral contraction, the cessation of the murmur is not attended by loss of the first sound. On the contrary, the heart, as it were, regains the first sound, which for a time had been merged in the valvular murmur. It is then probable, that the valvular sound having been eliminated, the great source of the systolic sound is the contraction of the left ventricle.

Finally. There is a form of morbid muscular action not described by Skoda, in which the voluntary muscles are liable to extraordinary and sudden contractions, so abrupt and well defined as to produce a succession of sharp and distinctly marked sounds of singular intensity. Minor degrees of this disease are not uncommon. Thus we find the phenomena in question on examining with the stethoscope the supra-spinous and acromial regions in young persons, in which a nervous condition simulates phthisis, and the rustling sounds thus produced are often mistaken for tuberculous râles. But in a case which I have frequently examined, the patient, a young man, can at will produce a succession of sounds from the left shoulder, so loud and sharp that they may be compared to the sounds of squibs or the cracking of a whip. When the ear is placed on the shoulder, the sharpness of the sounds becomes painful. He has also the power of producing sounds, evidently of the same nature, at the epigastrium and along the insertion of the left ala of the diaphragm.

All these facts make it probable that muscular action may have, occasionally at least, some part in the production, if not of both sounds of the heart, at least of the first sound.

CHAPTER III.

DISEASE OF THE MUSCULAR STRUCTURES OF THE HEART.

THE parietes of the heart are probably liable to all the vital and organic changes observed in the muscular structures. They may exhibit hypertrophy or atrophy, fatty degeneration, and heterologous deposits, or become the seat of changes which are secondary to various essential diseases, but especially typhus fever. Their functional diseases, also, seem analogous to those of muscles in general, as we observe augmented or diminished contractility, irregular action, and even a spastic state.

Further, we find that a weakened or paralyzed condition is produced by the effects of irritation of structures with which they are in connexion; this, as has been already noticed, may be the cause of death in pericarditis; and there appear reasons for believing that a purely nervous paralysis may affect one or more of the cavities of the heart.

Again, the muscles of the heart may be the seat of inflammation (myocarditis,) and although this condition is rare, it is probably more frequent than inflammation of the voluntary muscles.

It is still to be determined whether the cavities of the heart are liable to change from mechanical causes alone; whether dilatation, for example, is a purely mechanical result of obstruction to the exit of the blood, or whether for its production in valvular disease there is required not only obstruction, but a weakened condition of the heart.

As the symptoms of valvular diseases are really those of alterations of the cavities of the heart, we may now properly examine the latter class of affections, premising that although dilatation and hypertrophy so frequently co-exist with alterations of the valves, yet that they occur either as independent affections, or with an amount of valvular disease so insignificant as to constitute an accidental and unnecessary complication; we must, however, note a class of cases which, with the symptoms of dilatation, present the acoustic signs of valvular lesion, arising not from disease of the valves themselves, but from this, that the enlargement of the orifices renders the valves inadequate. Of this the case already given (see page 184) is an example; such a condition is more likely to occur in the case of dilatation without hypertrophy, for in the latter case the valves themselves become extended and enlarged, partaking, as it were, in the general increase of the heart.[1]

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[1] To determine the existence of dilatation or hypertrophy of the heart on dissection is often a matter of great difficulty to physicians, a circumstance not to be wondered at when it is considered that so few have examined the actual or normal dimensions of the organ. Among the investigators who have taken up this subject, the first place must be given to Bizot, whose measurements of the heart, under the various circumstances of age, sex, and disease, are more numerous, and probably more accurate than those of any preceding observer. His Memoir, entitled *Récherches sur le Cœur et le Système Artériel chez l'homme, par J. Bizot* (*de Genève*,) *Mémoires de la Société Médicale d'observation de Paris*, 1886, illustrates the value of the numerical method in determining questions of normal and pathological anatomy. The following mode was adopted in measuring the heart:—the breadth at the base was measured near to the union of the auricles and ventricles, while the length was represented by a line arising at the apex of the organ, and falling perpendicularly on its base. The thickness was also ascertained; the left ventricle was then opened by an incision along the rounded margin of the heart from the apex to the base, and prolonged to the aortic orifice; and, in order to convert the ventricle into a plane surface, the auriculo-ventricular orifice was divided. The length of the line passing by the convex and adherent margins of the sigmoid valves, and terminating at the two incised edges of the wall of the ventricle, gave the circumference of the base of the ventricular cavity; and a second line, drawn from the summit of the cavity and falling at right angles on the first, measured its height. In measuring the thickness, three points were taken, namely: 1. Towards the base, at six lines from the origin of the fleshy fibres. 2. At the point of greatest thickness, which is found near the union of the lower to the middle third of the ventricle, measuring from the base. 3. At a point four lines above the apex of the heart. The same points were chosen for the measurement of the septum. The right ventricle was measured as follows: it was divided from the base to the apex on its posterior portion, following the line of union with the inter-ventricular septum; another incision was made at its anterior surface, starting from the pulmonary artery, and following the line of the septum. The ventricle was thus divided into two portions, one belonging properly to it, the other constituted by the ventricular face of the septum. The different measurements were made as in the case of the left ventricle, care being taken to add the measurements of the two separate portions; and, avoiding in the measurement of the base to include the extent of the auriculo-ventricular orifice, and

DILATATION OF THE HEART.

The occurrence of an uncomplicated dilatation of the heart must be considered as one of extreme rarity. In most instances dilatation of the cavities is met with under two conditions:—

1. In connexion with valvular disease.

that of the pulmonary artery. The thickness was taken in the same way as in the left ventricle. M. Bizot has not published the measurements of the auricles: the dimensions of the arterial openings were ascertained by taking their circumference at the free border of the sigmoid valves and those of the auriculo-ventricular openings along the line of adhesion of the mitral and of the tricuspid valves; finally, the arteries, having been divided so as to form a plane surface, were measured at their origins, at their middle portions, and at their terminations. These researches were made upon 157 subjects, of all sexes, every possible care being taken to avoid error; the dimensions of the entire heart and its different portions being statistically studied according to age, sex, the height of the individual, and, finally, under the influence of disease.

These investigations were all conducted according to the numerical method of Louis, a method which, whatever may be its dangers and difficulties as applied to the determination of the value of remedial measures, is admirably adapted for the settlement of many questions of normal and pathological anatomy; yet even in this latter department we cannot join with many advocates of the system in decrying the value of preceding investigations because they were not based on the numerical method; such a course, in fact, is to ignore the labours of all those investigators whose works, from the sixteenth century down, have made medicine a science. To the illustrious author of the numerical method these observations will not, of course, apply; yet the history of every doctrine shows us that the reputation of the master may be compromised by the zeal of ardent but inexperienced disciples.

The following measurements, taken from the memoir of Bizot, are given by Hasse. But as in the translation of his book by Dr. Swaine the French measures are adhered to, it became desirable that the Parisian inch and line should be reduced to the English standard. My friend Dr. Moore has kindly furnished me with the following Table, in which Bizot's results are expressed in English measures.

In subjects between the thirtieth and forty-ninth year, the heart presents (according to Bizot:)—

	English inches.	English inches.
	IN MEN.	IN WOMEN.
A length of	3.8299	3.6478
Breadth,	4.2480	3.9104
Depth,	1.5250	1.2563
Length of left ventricle,	2.6176	2.8363
Breadth of ditto,	4.7218	4.1053
Length of right ventricle,	3.3357	2.9731

2. As one of a group of lesions, in which organic and functional disease of the heart, lungs, liver, and kidneys co-exist. There is an asthenic and often a gouty condition of the system. So great is the frequency of cases which may be placed in this category, that we shall devote some space to their consideration.

The leading characters of a large number of cases of dilatation of the heart are as follows:—

1. Organic change of the valves is rare, and when met with it is inconstant in its seat, nature, and amount, and incompetent to explain either the symptoms or the condition of the heart.

2. There may be from dilatation of the ventricles such an enlargement of the orifices as that the valves become incompetent to close the openings. It is to be doubted whether in cases of this kind we see the actual extension and enlargement of the auriculo-ventricular valves, which are frequently observed in dilatation with hypertrophy of the heart.

3. The parietes of the heart are thinned, and in many cases loaded with fat. In some, too, the substance of the organ is in an early stage of fatty degeneration.[1]

4. This disease is commonly met with in connexion with chronic bronchitis, and the patient is liable to attacks of cardiac asthma. Hepatic congestion, also, is common, and we may fre-

	English inches. IN MEN.	English inches. IN WOMEN.
Breadth of ditto,	7.4090	6.8047
Thickness of the walls of the left ventricle at the base,	0.4324	0.3650
Do. at the middle,	0.4520	0.3996
Do. near the apex,	0.3165	0.2861
Thickness of the septum of the ventricles at the middle,	0.4362	0.3913
Thickness of the walls of the right ventricle at the base,	0.1640	0.1512
Do. at the middle,	0.1158	0.1101
Do. near the apex,	0.0868	0.0822
Width of the left auriculo-ventricular orifice,	4.2987	3.6100
Do. of the right,	4.8145	4.1867
Width of the origin of the aorta (above the valves,)	2.7412	2.4962
Do. of the pulmonary artery,	2.7991	2.6047

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[1] The researches of Drs. Paget and Ormerod should be consulted on this subject.—London Medical Gazette.

quently observe varying enlargement of the liver corresponding to each attack of pulmonary congestion.

5. This disease, which frequently terminates in general dropsy, is often met with in connexion with a gouty habit in persons advanced in life, and whose systems have been exhausted by over-fatigue or undue depletion.

The leading characteristics of this affection are those which indicate a weakened condition of the heart. The pulse is permanently irregular, unequal, weak, and generally small; and the patient suffers from dyspnœa, with occasional attacks of orthopnœa, which are commonly induced by cold or fatigue, or are ushered in by diminished secretion from the kidneys. It is under these circumstances that the already enlarged liver exhibits a rapid increase of tumefaction, in a few hours descending far into the abdomen, yet on the subsidence of the attack returning to its ordinary volume, when it may be felt as a flat and indolent tumour extending for an inch or more below the false ribs. This phenomenon has probably a double origin, and may arise from the combined effects of enlargement and of displacement. The enlargement is caused by the distention of the hepatic veins, and the displacement by the tumefaction of the lung, which, as it is generally emphysematous, is so distended at each new attack as to produce an eccentric displacement of the ribs, mediastinum, and diaphragm, but resumes its former dimensions when the paroxysm has subsided.[1]

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[1] The idea of Serres, that disease in man not only repeats the embryonic state of the viscera, but may actually reproduce the normal state of organs in the lower animals ("Recherches d'Anatomie Transcendente et Pathologique par M. Serres; Paris, 1833,) may be referred to in considering this condition of the liver. On the great doctrine of Serres, that pathological anatomy is not a science of exceptions, which is after all the same as that of Broussais, though expressed in different words (see his "Commentaires sur les Propositions de la Pathologie,") I have already expressed my opinion in a review of Serres's work in the Dublin Medical Journal, First Series, vols. ii. and iii. But the close analogy between the condition of the liver with which we are now occupied and that of the diving animals is very remarkable. On this subject Professor R. W. Smith has the following observations, with reference to the case of Mr. Colles, in which the varying enlargement of the liver was a prominent symptom:—

"This interesting phenomenon was long since observed by Andral, in cases of cardiac disease, obstructing the course of the venous blood in the lungs, and it affords another proof that the functions of the liver are supplemental to those of

The physical signs observed in this affection are exactly those which would result from weakness and dilatation of the heart; but it is to be noted that our experience of these cases is drawn from studying the disease when complicated with affections of the lung and liver. No case of simple dilatation has been observed by me, but I am not disposed to deny the possibility of such an occurrence.

There is a great similarity in the physical signs in these cases. We observe that the sounds of the heart are often so affected as to make it difficult to distinguish between the first and second, a difficulty increased by their shortness and the irregularity and rapidity of the action of the heart. Indeed, it is frequently no easy matter to analyze the action of the organ; generally, the

the lungs; the evidences of this fact derived from observing the condition of the fœtal liver, before the lungs are called into action, its state in animals with vesicular lungs, incapable of aerating and decarbonizing the blood perfectly, as well as in examples of open foramen ovale, are so well known to physiologists that it is unnecessary to do more than refer to them. The phenomenon in question is, however, best elucidated by the observations made by the late Dr. Houston, on the circulating organs in diving animals: he has shown, that in animals which are capable of bearing submersion for a long period, as diving birds, the porpoise, the seal, the otter, &c., the veins connected with the liver are dilated into enormous reservoirs, which serve as a temporary resting-place for the blood, when stopped in its free course, during the obstruction to respiration which occurs in the act of diving; and this provision or reservoir is much enlarged, and most generally extended throughout the venous system of the body, in those animals which are capable of enduring submersion for the longest period: in the others, whose submersion is only occasional, and that but for a short period at a time, when diving for their prey in shallow, inland water, the hepatic veins alone are dilated into receptacles for the blood retarded in its course; but in the seal and in the porpoise, who frequent deep waters, and whose submersion is more prolonged, the provision of a reservoir is extended throughout the greater part of the venous system of the body. These ingenious observations of Dr. Houston appear to me to admit of being legitimately made use of to explain the occurrence of the occasional and temporary enlargement of the liver, in cases such as that under consideration; they serve to prove that it is a means of diminishing the dangers arising from pulmonary congestion, and a provision for retarding the circulation of venous blood through the system, while respiration is seriously obstructed, and the lungs incapable of aerating the blood so as to maintain life. In conclusion, I have only to observe, that the bilious tinge of the skin and the formation of gall-stones in this interesting case are most probably to be referred to the obstruction of the pulmonary circulation. The observations of Tiedemann and Gmelin tend to prove that, in such cases, the secretion of bile becomes more abundant."—*Reports of the Pathological Society*, 1843.

sounds are louder in the lower sternal than in the mammary region, and this condition, which represents the permanent state of the heart, is aggravated in all its characters during each of the paroxysms of dyspnœa to which these patients are liable. At such times the impulse of the heart is often increased. It is stated by authors that no præcordial fulness exists in this disease: but while we admit this statement, we cannot hold that the want of præcordial fulness is a diagnostic between this affection and dilatation with hypertrophy, inasmuch as in the latter disease this physical sign is often wanting.

As a general rule, we do not observe valvular murmur in this affection, at least it rarely occurs in the special case under consideration; yet we are not justified in declaring that simple dilatation is never attended by murmur; nor again that where murmur does exist, it is to be attributed, as Dr. Walshe believes, to an enlargement of the orifices consequent on the dilatation of the cavities. I have observed in a case of this kind that the murmur which existed in the earlier periods of the disease disappeared during the last years of the patient's life. This murmur had the usual characters of a mitral murmur, and dissection afforded no explanation either of its appearance or disappearance.

There is not only a great similarity in the symptoms and signs of this combination of diseases, but also in the mode of death. Each attack or paroxysm, as it were, places the patient in a worse position, until at length the lungs become congested, and death by asphyxia closes the scene. In the case of Mr. Colles, extensive solidification of the lungs took place shortly before death, attended with bronchial respiration and dry friction sound. Yet the appearances on dissection were rather those of splenization than of hepatization.[1]

Although these cases are to be met with every day, especially in private practice, we still observe that physicians differ as to their nature. One holds that the liver is the organ in fault; another, that the disease is in the valves of the heart; a third believes that the symptoms are those of hydrothorax, from disease of the kidney; while a fourth sees nothing but misplaced gout. Each of them may be said to be in one sense right, all of them in another sense wrong. That the heart, liver and lung are in fault, in most of these cases is certain; that the kidney is functionally affected, and the gouty condition present, is commonly true. But we must learn to look fairly at the entire case, and not dwell on its separate phenomena.

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[1] Observations on the Case of the late Abraham Colles, M. D., &c. By William Stokes, M. D. Dublin Quarterly Journal of Medical Science, vol. i. p. 303.

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In a clinical point of view these cases form one of a group of diseases which may be classed as examples of weakness of the heart. For although they differ in their special signs and symptoms, and, above all, in their history and accompanying circumstances, yet they agree in exhibiting a diminished force, especially of the ventricles.

In the case of hepatic complication we observe that mercurial action produces a singularly beneficial effect. This is not easy to explain. We do not know in many of these cases whether the hepatic or cardiac disorder has had the initiative. This much is certain, that, the combined disease being once established, a mutual re-action takes place between the heart and the liver, so that whatever influences one of these organs, will produce a new disturbance in the other.

As an illustration of what has been now said on this form of disease of the heart, which of the examples of dilatation, is unquestionably the most frequent, I will give the case of my venerated friend and teacher, the late Mr. Colles, who so long filled the Chair of Surgery in the Royal College of Surgeons in Ireland. The case of this remarkable man and eminent surgeon was published by me in 1844, but I believe that in inserting it in this work, though in an abridged form, I shall be acting in accordance with the expressed desire of Mr. Colles, that the history of his case should be, as far as possible, made available for the advancement of medicine.

Mr. Colles, as he advanced in life, experienced frequent attacks of gout in its ordinary form, and from about the year 1834 was the subject of a chronic bronchitis, with occasional exacerbations of the disease in an acute form. During these attacks the prominent symptoms were dyspnœa and palpitation, and the treatment adopted was to employ small general bleedings, followed by the use of blue pill and Dover's powder. He was occasionally

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liable to erysipelas of the face in a mild form, and it was found that both the erysipelatous and gouty attacks were attended with a suspension or diminution of the affection of the chest.

In this state of health Mr. Colles continued for about six years, during which time he hardly ever intermitted those laborious duties which all who rise to eminence in the profession of medicine must undertake and bear as they best may. In the spring of 1840, however, the first symptoms of a yielding of the system took place, and in a sudden manner. Mr. Colles had retired to bed, feeling as well as usual, but during the night was seized with a paroxysm of cardiac asthma. He described the sensation of impending suffocation at the commencement of the attack as being dreadful. He remained in a state of orthopnœa during the night, with wheezing respiration. In the morning the pulse was rapid, irregular, and unequal,—a condition to which the action of the heart corresponded. The chest was clear on percussion. A fit of gout in the lower extremities soon supervened, but on its disappearance the legs remained unusually œdematous.

At this period of his case the heart presented the following physical signs:—The impulse was feeble, irregular, and rapid, and the organ seemed to impinge over a large surface. So irregular and rapid was the action of the heart, that the analysis of the sounds was a matter of great difficulty, the first occasionally resembling the second sound, and *vice versâ*. There was no valvular murmur, nor any unusual pulsation or thrill in the arteries.

A few months having elapsed, Mr. Colles was recommended to try the effect of change of air and travel, with the double view of obtaining some advantage from the effects of a new climate, and the benefit of rest from his professional exertions. He proceeded to Switzerland, where his health was so greatly improved that on one occasion he found himself able to walk up hill for a considerable distance. This restoration of his former powers of exertion afforded him great happiness. Some time, however, after his return to Dublin, his old attacks returned. I saw him after an interval of several months, and for the first time observed that the liver was permanently enlarged, forming a smooth, flat tumour. He continued to suffer, from time to time, from paroxysms of dyspnœa, which were generally preceded by diminution in the secretion of the kidneys. During these attacks, which generally lasted for several days, the irregularity of the heart and the præcordial distress increased, until orthopnœa was established. The kidneys acted scantily, and no copious sediment appeared in the urine. On each attack the tumefaction of the liver increased with great rapidity, but this condition as rapidly subsided with the improvement in the symptoms. No relief was ever obtained until a free action of the kidneys was established; but it was found that this could only be effected by the use of mercury followed by diuretics. On several occasions the diuretic treatment, not preceded by mercury, was tried, but it always failed, so that the number of times in which a distinct course of mercury was employed was very great. To this remedy, in a great degree, must the prolongation of Mr. Colles's life be attributed: for, on various occasions, the symptoms had gone so far as to cause complete orthopnœa, with unusual anasarca, and alarming pulmonary congestion.

In this condition of intervals of comparatively good health, while the attack came on once in about every five weeks, Mr. Colles continued till the summer of 1843, when, after a mild course of mercury, continued for many weeks, Mr. Colles regained a state of health to which he had been long a stranger. His appearance improved, he even gained flesh, and had an excellent appetite. Another bad attack supervened in the early part of the autumn, but it yielded to the usual treatment. But this was the last time that the system responded to medicine. In October a new invasion of the disease set in, having precisely the characters of the former attacks; and for the first time the mercurial treatment failed. The anasarca increased, and the occurrence of a congestion of both lungs, so great as to cause general dulness and bronchial respiration, was the immediate forerunner of death, which took place on the 1st of December, 1843.[1]

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[1] Were I the biographer of Mr. Colles, I might enlarge on the many excellent qualities of his mind, on the independence of his character, his boldness of thought, his warmth and largeness of heart, and his unquenchable zeal in the practice and the teachings of his profession. It is only when we lose a great possession that we are able to estimate its full value. But it is a privilege allowed to the good and wise, that their example, which in one sense is their spirit, remains after them. Clear in his convictions as to what was right, and steadfast to do and to

The examination of the body was made by Professor R. W. Smith, in the presence of Sir Henry Marsh, Professor Harrison, and myself.

"The surface of the body generally was œdematous, but the swelling was greatest in the hands and feet; the skin was slightly tinged with jaundice. On opening the cavity of the thorax, it was observed that the costal cartilages had been converted into bone: when the sternum was removed the sac of the right pleura was found to contain about half a pint of dark-coloured serum, in which were suspended numerous flakes of lymph, which appeared to have been recently effused; the right lung was, throughout its whole extent, in a state of extreme congestion, and at its base was expanded into two large globular tumours, each about the size of an orange, heavy and dark-coloured, though obviously of an emphysematous character: when divided through their centre, they were found to contain not only air, but also a considerable quantity of dark blood, of a venous character, producing an appearance very like that of the interior of the spleen. When the blood was washed away the surface of the section presented a highly vesicu-

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teach only that which he thought was right, Mr. Colles gave to Irish surgery a great impetus, and a lustre which it cannot lose. From an early period of the illness which terminated his existence, Mr. Colles was in the habit of speaking calmly and freely on its nature to me and his other medical friends, and of giving his views as to its probable termination. So far back as the summer of 1842, he observed to me that a time must soon arrive when those remedies which had so often succeeded must fail; and he directed that a careful examination of his remains should be made by Professor Smith, in the presence of his medical attendants. "I think," he said to me, "that this examination will add to our knowledge, and I know that the dissection will be made with accuracy and the result be truly given." He subsequently wrote the following letter to Professor Harrison:—

"*October* 22, 1842.

"MY DEAR ROBERT,—I think it may be of some benefit, not only to my own family, but to society at large, to ascertain by examination the exact seat and nature of my last disease. I am sure you will grant my request, that you will see that this be *carefully* and *early* done. The parts to which I would direct particular attention are the heart and the lungs, a small hernia immediately above the umbilicus, and the swelling of the right hypochondrium.

"From the similarity of the Rev. P. Roe's case with mine, I suspect that there is some connexion between this swelling of the hypochondrium and the diseased state of the heart.

"Yours truly, dear Robert,

"A. COLLES."

lar aspect; the cells were large and very irregular. The entire of this congested lung, with the exception of a small portion at the apex, was more or less solid, but did not present any of the characters which distinguish solidification, the result of pneumonia; it did not break down under moderate pressure; the solid feel which it possessed arose from its extremely congested state.

The sac of the left pleura was obliterated throughout its whole extent by organized adhesions, which were evidently of very long duration; the left side of the chest was contracted; the lung, smaller than natural, gorged with blood, and sunk back towards the spine, yielded to and broke down under a very gentle pressure: it presented a purplish red colour, did not crepitate anywhere, and resembled closely the appearance of the spleen, when under the influence of decomposition; the bronchial glands, in the posterior mediastinum, were enlarged, and contained calcareous matter. There was no effusion into the sac of the pericardium, nor any adhesion between its opposed surfaces. The heart was much larger than natural, but not proportionably increased in weight; its left cavities were collapsed and flaccid, while those of the right side were distended with dark blood, especially the auricle. The surface of the organ was of a pale brown colour; the quantity of fat upon it was much greater than natural; its muscular tissue, pale, soft, and greasy, was easily ruptured. The left ventricle did not contain any blood; its cavity was remarkably large, but there was no hypertrophy of its parietes: it presented an example of great passive dilatation; the left auricle was also empty; the auriculo-ventricular openings were natural; and the same may be said of the aortic orifice—at the attached margin of one of the valves there was a small particle of calcareous matter; it was not as large as the head of an ordinary-sized pin, and in no way interfered with the due exercise of the functions of the valve; water poured into the cavity of the aorta did not enter the ventricle; the lining membrane of the aorta was stained of a deep red colour, and several atheromatous depositions were observed beneath it; numerous globules of oil were seen floating upon the surface of the blood, which collected in the chest during the examination of the heart. The sac of the peritoneum contained about a quart of fluid; the liver, though not much enlarged, extended below the margin of the ribs; it was of an exceedingly dark mahogany colour, presenting a tumid and swollen aspect, and a rough and granular surface. When a section was made through it, the dilated veins poured out copious streams of exceedingly dark blood; the gall-bladder contained thirty moderate-sized gall-stones. Upon the right side of the umbilicus there existed traces of a small hernia, which Mr. Colles had requested might be examined; when a section was made through the kidney, globules of oil flowed with the blood; the remainder of the urinary apparatus and prostate gland were quite healthy."[1]

Let us now take a general view of the symptoms in these cases. They are commonly held to proceed from contraction of the mitral orifice. At first sight it would appear a matter of little consequence, as to whether the disease was a valvular affection, or was seated in the muscular apparatus. Yet great errors in practice may result from an erroneous view of the case being taken. The patient is forbidden stimulants to which he has, perhaps, been accustomed. He is put on a spare diet, digitalis is used, and all active exertion inhibited from the fear of its causing sudden death, an apprehension which, when conveyed to the patient's mind, produces the worst effects.

This disease presents itself in a twofold aspect, namely, in its stages of quiescence and of paroxysmal aggravation.

In the intervals of the exacerbations we may find our patient, to all external appearance, in a good state of health. He eats, drinks, and sleeps well, and not unfrequently is able to fulfil his ordinary avocations in life, so far as these can be performed without great muscular effort. If he be a professional or mercantile man, he can attend effectively to his duties. His head is clear, and his nervous energies unaffected. He may have a slight degree of œdema of the lower extremities, and a chronic cough, but this is attributed to gout, and to an habitual bronchitis. We find, however, that his powers of ascending any elevation are much diminished, and his physician observes that the pulse is permanently small, weak, irregular, and intermitting; and if an examination of the abdomen be made with care the liver can be felt flat and extending considerably below the margin of the ribs;

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[1] Dublin Quarterly Journal of Medical Science, Second Series, vol. i. 1846.

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yet the patient feels in good health, he has no jaundice, and is able to enjoy society.

The exacerbation generally comes on in connexion with an increase of the bronchial affection, till severe orthopnœa is induced, and it begins with diminution of the renal secretion, followed by the most extreme cardiac and pulmonary suffering.

The dyspnœa and diminution of urine are the principal symptoms. One or two dreadful paroxysms of cardiac asthma may occur, while at intervals, an apparently increasing bronchial effusion threatens the life of the patient. The suppression of urine is extremely rapid, so that in two or three days the kidneys secrete but a few ounces of fluid. The patient is tormented by the difficulty of breathing and the apprehension of approaching death, and the pulse becomes more rapid, more feeble, and irregular, while the action of the heart is such as almost to defy any stethoscopic analysis. The rhythm is altogether disturbed, and a protracted observation is necessary to determine which is the first and which the second sound of the heart. The respiration is laborious, wheezing, or even rattling; the chest sounds clear on percussion, but extensive sonoro-mucous rattle is largely developed, while the signs of congestion, or even œdema, of the pulmonary structure are commonly to be observed in the postero-inferior portions of both lungs. No signs, however, of liquid effusion into any of the serous cavities are discoverable, though the patient presents all the symptoms of hydrothorax as laid down in nosological works.

The hepatic complication is of great importance, and presents some singularly striking phenomena. Without fever or gastro-intestinal inflammation, the liver is observed to enlarge often to such an extent that the tumour may advance below the umbilicus. This augmentation occurs with great rapidity, but is unattended with any signs or symptoms of hepatic inflammation, and it subsides to a greater or less degree when the state of paroxysmal suffering has been subdued. Andral has noticed this singular augmentation of the liver, which is often as remarkable and recognisable as that of the enlargement of the spleen in ague. The tumour is flat, and either painless on pressure or very slightly tender. With each paroxysm of the disease the hepatic tumour seems to gain a slight permanent increase; but the alternation of its enlargement and diminution, corresponding to each attack of the disease, forces the idea on the mind of the observer that the organ is in an erectile condition.

One of the most remarkable circumstances in this curious combination of symptoms is the suppression of the renal secretion, and the subsidence of at least the aggravated symptoms of the attack on its restoration. There is no reason whatever to believe that the kidney is the seat of organic disease.

It is difficult or impossible, in the present state of our anatomical knowledge, to explain the phenomena of this disease. The morbid state of the heart, consisting in its weakness, dilatation, and irregular action, and the permanently enlarged, though indolent condition of the liver, may be taken as the constant characteristics, while the exacerbations of the bronchitis on the one hand, and the suspension of the renal secretion on the other, are the accidents commonly attendant on the paroxysm of the disease. We may suppose that either of these affections, or both of them concurrently, by inducing an accumulation of blood at the right side of the heart, may cause the paroxysm of cardiac suffering, attended by anasarca, owing to the general congestion of the venous system; and, on the other hand, by overloading the venæ cavæ hepaticæ, may induce a passive enlargement of the liver. We may suppose that the repetition of these attacks establishes a permanent hypertrophy of the latter organ, which in its turn becomes an exciting cause of disease, so that the cardiac and hepatic affections are reciprocally cause and effect; and that such is the case appears probable from the history of them in many instances.

SIMPLE UNCOMPLICATED DILATATION OF THE HEART.

I have clearly expressed my opinion that this disease is one of extreme rarity, and as I cannot produce any original observations of such a condition, it appears better to state generally, that the diagnosis is to be drawn more from theoretical considerations than from observed facts. It depends on the existence of signs of an enlarged and at the same time weakened heart; and the signs vary according as the dilatation predominates in the right or left side of the organ. To declare that we can distinguish between a dilatation with thinning of the parietes of the heart, and that form of enlargement where the capacity of the cavities, as well as the thickness of their walls, is increased, while the force of the organ is not augmented,—is to state what is not warranted by clinical experience.

Excluding all considerations of valvular obstruction, or of disease of the lung or liver, it may be laid down that the dilated state of the heart is more often seen in the right than the left cavities; and even under the circumstances now specified, it is a rare affection: that is to say, if we exclude that form of which an illustration is furnished by the case of Mr. Colles, a form in which not only disease of the liver and lung add their quota to the group of symptoms, but where the gouty condition of the entire system is an important element, the occurrence of simple uncomplicated dilatation of the heart, considered without reference to any muscular degeneration on the one hand, or valvular obstruction on the other, is so rare an affection that, while we do not deny the possibility of its occurrence, we must admit that there is little, if any, clinical observation which would establish its diagnosis.

The following should be the theoretical diagnostics of such an affection:—

1. Increase of the area of dulness over the heart.
2. Feebleness of impulse.
3. Feebleness and smallness of pulse.
4. Feebleness of the sounds of the heart.
5. Absence of true valvular murmur.

To these diagnostics may be added the following:—That the patient may be liable to cerebral attacks, resulting either from deficient supply to the brain or from nervous congestion; and that he may exhibit symptoms of dyspnœa on exertion, and the signs of an overloaded right ventricle, as shown by jugular pulsation, and perhaps an engorged state of the liver. Finally, a dropsical tendency will probably be manifested.

In speaking of the differential diagnosis between dilatation of the heart and the combination of dilatation with hypertrophy, Laennec has stated, that a certain clearness or sharpness of sounds attends the dilated state. This can hardly be admitted, unless we suppose a case in which there are the combined conditions of thinning of the parietes, with an increased vivacity or force of the muscular contraction. Whether such a state of the heart ever exists is very doubtful; and it is not improbable that in the mind of Laennec the connexion between clearness of sound and thinning of the parietes of the ventricles was but a corollary to his doctrine, that the second, or clear sound, was produced by auricular contraction. It is true, that in certain cases of great thickening of the heart a dull sound is produced; and also, that in some examples of dilatation the sounds of the heart have a sharp or flapping character; but there is really no evidence to show that these phenomena depend on any mechanical condition; and it is more consistent with the present state of our knowledge to attribute them to a deficient or increased contractile power. Certain it is, that the most remarkable examples of augmented loudness of both sounds of the heart are to be met with in hysteria or other nervous affections where no mechanical change of the organ can be supposed to exist. On the other hand, it may be objected that the contraction of the right ventricle gives a clearer sound than that of the left. But we cannot as yet affirm that the sound of one ventricle can be distinguished from that of the other; and even if the fact be admitted, there may be other causes for the difference in sound.

Lastly, it is to be remarked, that although in theory we do not admit true valvular murmur as a sign of dilatation of the heart, yet, on the other hand, when the dilatation of the cavities is carried beyond a certain point, valvular insufficiency may result, and then a murmur, as in the case already given, is produced; which, though having its origin in the valvular orifices, yet does not proceed from valvular disease.

DILATATION WITH HYPERTROPHY OF THE HEART.

This condition, so common in cases of valvular obstruction or imperfection, is yet, in its simple form, of very great rarity. Indeed, in an elementary work on practical medicine, its consideration might well be omitted; for in a great proportion of cases of enlarged and thickened hearts, valvular disease in the mitral or aortic opening, or in both simultaneously, is to be met with.

However, as a few examples of the uncomplicated affection have been recorded, we may, as in the case of dilatation, state the theoretical diagnosis.

Presuming that the contractile force of the heart is at least not below the normal state, the following signs will be observed:—

1. Increase of dulness, generally commensurate with the extent of the organ.

2. Increase of the force of the impulse at the side, and of the extent of surface over which this impulse can be perceived. This extension of the area of impulse is one of the best-marked signs of enlargement of the heart; and a moderate experience will enable us to distinguish between the impulse communicated by the surface of an actually enlarged heart and the sensation given in simple excitement of the organ. To this point we shall presently return.

3. The sounds are generally augmented, and commonly unattended by murmur. Occasionally we meet cases in which the first sound is attended with the ringing character; but as this phenomenon occurs in cases of ordinary nervous excitement, and is absent in dilatation without hypertrophy, it must be referred to an extreme activity of muscular contraction rather than to the dilatation, or even thickening of the ventricle.

But it is often found that a greatly enlarged heart may exist without much augmentation of sound or of impulse. The organ does not contract with vivacity; and hence, though by the hand placed over the præcordial region we recognise a deep and extended pulsation, we find this pulsation feeble and wanting in localization. It is not uncommon on dissection to find the heart much more enlarged than could have been expected from the sounds, impulse, or pulse, as observed even for a considerable time before death.

In such cases there is probably more or less of fatty degeneration, especially of that kind in which the fat globules are interstitially deposited in the fibre. It may be also that there is a true deficiency of the nervous power; or, lastly, that the organ, from its very bulk, has not sufficient room for full expansion, and consequently, cannot put forth its entire contractile power. Considered practically, we gain but little from examining the subject of hypertrophy of the heart when occurring independently of obstruction or dilatation of the valvular openings. Nor has medicine been much advanced by our attempts to study the signs of the lesion in this or that cavity; for though the signs of disease in either ventricle may be declared from *à priori* reasoning, we are taught by practical medicine that hypertrophy, with or without dilatation, is rarely confined to a single cavity.

DILATATION WITH OR WITHOUT HYPERTROPHY OF THE AURICLES.

Although in most cases of dilatation of the heart we find the auricles, as well as the ventricles, engaged, yet our knowledge of the disease as affecting the former cavities is very limited. Fortunately, this is not of much consequence to practical medicine. We do not yet know of any signs or symptoms by which the dilatation of one or both auricles could be directly determined. The existence of such a state will be probable when we find signs of enlargement of the heart, and especially if there be a contraction of the mitral orifice. Under these circumstances both auricles become engaged, and the left exhibits, as Dr. Adams has shown, opacity of its lining membrane, and the enlarged openings of the pulmonary veins. The circle of diseased actions is completed by the occurrence of pulmonary congestion, and of dilatation of the right ventricle and auricle.

I have already remarked on the difficulty which the anatomical position of the left auricle offers in any attempt to discover its enlargement by physical signs. This condition can only be inferred when we find the signs and symptoms of disease of the right cavities succeeding to narrowing of the mitral orifice. Let us suppose, for example, a patient who, for a certain period, presented a mitral murmur, but had no symptom indicative of pulmonary disease or overloading of the heart:—now, if in such a case the heart's action should become permanently irregular,—if hæmoptysis took place,—if the patient suffered from dyspnœa on exertion, while the jugular veins pulsated, and the apex of the heart could be felt beating in the epigastrium;—we might safely conclude that the left auricle was in a state of dilatation and, probably, hypertrophy; that the pulmonary veins were enlarged; and, finally, that the obstructive process had affected the right cavities. The diagnosis, however, will be inferential, for such a case as I have given, of dulness probably proceeding from enlargement of the left auricle,[1] must be one of great rarity; and we have no demonstrative proof that the explanation of its attending circumstances was correct. I have already expressed my opinion as to the uncertainty of the diagnosis which assumes that direct physical signs attend this condition.

We might, perhaps, go a step further, and say that hypertrophy, as well as dilatation of the auricle, may be expected in cases of narrowing of the mitral opening, while a simply dilated condition would probably occur in its enlarged and patulous state, of which the case by Mr. Fleming furnishes an example.

But the anatomical relations of the right auricle render it more favourable for the application of direct diagnosis; and it is probably liable to greater distention than the left cavity. There is reason to believe that this dilatation, when carried to an extreme degree, may be attended by two remarkable physical signs, namely, dulness on percussion, and *a pulsation which is probably diastolic.*

Some years since, a man past middle age was admitted into the Meath Hospital, labouring under symptoms of disease of the heart and general venous obstruction. There was considerable dulness to the right of the sternum, unexplained by any disease of the lung or pleura. The heart was in its natural position; its action irregular, and rather feeble than otherwise. Between the second and fifth ribs, on the right side, and corresponding to the dulness, there was a deep-seated but most distinct diastolic pulsation. This was synchronous with the first sound of the heart; but I am now unable to say whether murmur was present. For some days I inclined to the opinion that the case was one of aneurism, as the signs closely resembled those which we had observed in a case of true aneurism of the aorta. But when I considered the rarity of true aneurism, and also, that in all the cases of this disease that I had witnessed, the circulation was but little disturbed, I concluded against such a diagnosis. The pulsation was clearly different from that of the ventricle, although synchronous with it. It extended over a large surface, and had precisely the characters perceived in an aneurismal tumour in which the pulsation is not energetic. It is difficult to express in words the character of this pulsation; but to the experienced clinical observer I shall be easily intelligible. After a few days the pulsation became less distinct, and the symptoms of pulmonary congestion more decided. The patient sank in about a fortnight after his admission. The aorta was found perfectly healthy throughout its entire course; the lungs were extremely congested, and had evidently been long affected by Laennec's emphysema. The right ventricle was dilated and somewhat hypertrophied; but the right auricle presented a most singular appearance when the chest was opened, resembling a vast purple tumour which concealed the whole of the anterior portion of the right lung. Its parietes were in many places extremely thin, while in others the fleshy columns, especially in the appendix, were hypertrophied. Its cavity contained more than a pound of fluid, but grumous blood.

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[1] See page 220.

The great size of the auricle furnishes an easy explanation of the dulness on percussion, for there was no effusion into the pleura or consolidation of the lung. The great interest of the case, however, consists in the occurrence of pulsation, which must be supposed to have been caused by the introduction of blood *per saltum* through the auriculo-ventricular opening at each contraction of the heart. In fact, the auricle had become an aneurism so far as its mechanical relations were concerned.

This fact seems to open up some new subjects for consideration with reference to the heart's action in disease. If the auricles may become the seat of a throb, as it were aneurismal, it may be inquired, whether such a condition would be possible in the ventricles. If it be admitted that the auricles act *per saltum*, one of the conditions of such an occurrence would always exist; and it would only be necessary that the ventricle should be in a state of great debility, unable to empty itself completely at each retraction, in order to obtain the conditions necessary for such an occurrence. I apprehend that such an action takes place in certain cases of fatty degeneration with dilatation of the left ventricle, for I have observed instances of this disease wherein the systolic sound was extremely feeble, yet in which the impulse was diffused and clearly diastolic, having a close resemblance to that produced in a true aneurism of the ascending aorta. The character of this impulse was altogether different from that produced by contraction of the ventricle. It was eccentric, and its great dissimilarity to the ordinary impulse in fatty hearts tends to confirm the idea that it was produced in the ventricle by the systole of the auricle.

We should expect that the aneurismal pulsation of the auricle would be more likely to occur in the right than in the left cavity, when we recollect the frequency of the reflex jugular pulsation, and the natural imperfection of the tricuspid valves.

Let us now sum up what has been said on dilatation of the heart with or without hypertrophy.

1. That dilatation of the whole heart, or of any corresponding pair of its cavities, or of any single cavity, considered as a purely local disease, is one of extreme rarity.

2. That while uncomplicated dilatation of the heart is so rarely met with, the opposite form is of common occurrence.

3. That the cases of complicated dilatation are of three kinds. In the one the complication is related to disease of the orifices; in a second form, to obstruction in organs remote from the heart; and in the third, it appears to arise from a debilitated state of the cardiac muscles themselves.

4. That in the last condition the nervous deficiency or weakness of the heart is often connected with an early stage of fatty transformation of the muscular fibres.

5. That in cases of complication with valvular disease, the dilatation of the cavities, and especially of the left ventricle, appears to be the effect of regurgitation rather than of mere obstruction to the exit of blood.

6. That dilatation of the heart, in its most common form, is met with as one of a triple group of local diseases, in which the heart, lungs, and liver appear to be affected.

7. That in many of these cases the local affections are themselves secondary to certain morbid states, of which the most common are a gouty diathesis in an enfeebled subject, the anæmic or scorbutic state, or some other form of cachexia.

8. That in this condition both the structure and functions of the lung are commonly deranged, and we meet with chronic bronchitis, dilated tubes and air-cells, and various degrees of pulmonary congestion.

9. That again the liver is the seat of deranged structure and

function. It is generally enlarged, and yet its volume is observed to increase with each exacerbation of the disease.

10. That this paroxysmal swelling of the liver may rapidly subside, leaving the organ in its former state of enlargement at the close of each exacerbation of disease.

11. That in this triple combination the patient is liable to paroxysms of cardiac asthma, in which the three organs show symptoms of extreme derangement; that of the heart, by increased irregularity, rapidity and force of action; that of the lung by lividity, dyspnœa, and augmented râle; and that of the liver, by a rapid increase of its bulk, even though the permanent condition of the organ be one of hypertrophy.

12. That derangement of function in any of these organs may induce a paroxysm of disease, and that it is frequently impossible to determine whether disturbance of the heart, lung, or liver, has been the exciting cause of the attack.

13. That we cannot accept the opinion of Laennec,—that the distinctive sign of dilatation is the clearness of sounds during the systole and diastole of the heart.

14. That the frequent combination of weakness with dilatation of the heart should lead us to expect a feebleness of the sounds, and this more especially when it is recollected that the weakness is seldom unconnected with an organic change.

15. That dilatation of the left auricle, attended with more or less of hypertrophy, may be expected to exist in cases of mitral disease with contraction.

16. That we are not yet justified in declaring that the dilatation of the left auricle is attended with any special physical sign. It is probable, however, that in one observed case the sign of dulness on the left side suddenly occurring and stretching from the base of the heart upwards, may have been induced by distention of the left auricle.

17. That distention of the right auricle has been found to be attended with dulness, and with a diastolic pulsation synchronous with that of the ventricles, so as to simulate aneurism of the aorta.

18. That there are some grounds for believing that a similar action may be produced in a ventricle, when its contractile force is much diminished and its capacity increased.

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19. That we are not in a position to declare why it is that in one case we have dilatation without thickening, and in another, dilatation with hypertrophy.

20. That dilatation with preservation of the natural thickness is to be considered a form of dilatation with hypertrophy.—(*Hypertrophie dilatatoire* of Forget.)

21. That dilatation with increase of thickness may be met with although no valvular disease exists.

22. That, however, it is most commonly seen in cases of valvular lesion.

23. That hypertrophy with dilatation of the left ventricle may arise on the one hand from the regurgitant disease of the aortic orifice; and on the other, from the permanently dilated condition of the mitral opening.

24. That hypertrophy and dilatation of the left auricle are met with in cases of mitral obstruction.

25. That hypertrophy and dilatation of the right cavities are seen to occur in cases of pulmonary congestion; but that in many cases the point of departure of the entire disease seems to be a contraction of the mitral opening.

In connexion with the subject of dilatation of the heart in general, we shall now notice the not unfrequent case of palpitation of the heart attended with enlargement of the thyroid gland and eye-balls. Yet, although some form of dilatation of the heart has been found in a few cases of this disease, we cannot but consider it as a special affection, in which the organic change is secondary to functional derangement.

INCREASED ACTION OF THE HEART AND OF THE ARTERIES OF THE NECK, FOLLOWED BY ENLARGEMENT OF THE THYROID GLAND AND EYE-BALLS.

The following are the important features of this disease:—

1. Increased force and rapidity of the heart's action, without fever, and of long continuance.

2. Excited action of the carotid and thyroid arteries.

3. Enlargement of the thyroid gland, varying with the force of the heart.

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4. Enlargement of the eye-balls, without any disease of the orbits or brain.

This affection is most commonly met with in women, but males are not exempt from it; and it may arise at various ages. I have seen it in a lady upwards of sixty years of age.

The point of departure of the disease is the heart, the action of which becomes rapid and occasionally tumultuous; and subsequently, after a period of time varying in different cases, we observe the enlargement of the thyroid gland and also of the eye-balls, attended with a pulsation of the whole neck, especially in its lateral portions, and in the seat of the thyroid gland itself. When this pulsation is examined, three causes are found to concur in its production, or, rather, there are three different kinds of pulsation. We have, first, the arterial pulsation simply; next, the diastolic throbbing of the gland; and lastly, a pulsating thrill in the gland and veins of the neck, which is similar to the thrill of an aneurismal varix.

The thyroid enlargement and pulsation appear to precede the increase of volume of the eye-balls. Dr. Graves mentions three cases of palpitation in females, in which the tumefaction of the gland, arising with each attack, and diminishing with its subsidence, was observed. In these cases the enlargement of the eyes had not yet occurred; but, doubtless, had the disease continued sufficiently long, that complication would have been produced.

Some years ago, when the disease was but little known, a case of this condition of the thyroid gland in a young woman was actually mistaken for aneurism, and a day appointed for performing the operation of tying the carotid artery. Happily, the true nature of the affection was discovered in time, and the patient was cured by the use of sedatives and the preparations of iodine. The tumour in this case was larger than a hen's egg, and somewhat flattened anteriorly. Its pulsations were violent, and over every part of its surface the thrill of aneurismal varix could be felt. This was attended with the sounds peculiar to this condition. The eyes had not become enlarged.

This disease of the thyroid differs in some respects from ordinary bronchocele. The liability to its production is in no way connected with any of the influences of soil or climate. The volume of the tumour is remarkably variable; but even in cases where the disease had existed for several years, it seldom attains to a large size. Dr. Graves indeed states that the tumour is never sufficiently large to cause deformity of the neck, but I have seen two cases in which considerable deformity existed. The same author observes, that it is distinguishable from bronchocele by its becoming stationary just at that period of its development when the growth of the latter usually begins to be accelerated. I have observed this arrest of the growth of the tumour in several cases; in one instance, to which I shall again allude, where the disease occurred in the male, the tumour became more solid, and the thrilling sensation and murmur disappeared from various points of the surface. After some years all thrill and murmur had disappeared; the tumour felt solid and nearly inelastic, while a large varicose vein, with thickened parietes, ran over the front of the tumour: in the course of this vein, and in no other situation, the murmur still existed.

The accompanying phenomena, referrible to the action of the heart, the arteries in the neck, and the peculiar condition of the eye-balls, will be sufficient to establish the diagnosis.

But although these cases, which have so strong a generic resemblance, differ in their history and accompanying conditions from those of ordinary bronchocele, we cannot, without risk of error, describe them as examples of a perfectly distinct disease. The remarkable preponderance of both forms of the affection in females, at least in this country, is important, and if to this be added, that no instance has been observed of the affection we are now describing occurring before puberty,—that the structures engaged in both affections appear to be the same,—that in many cases of ordinary goitre, hysterical paroxysms, or uterine derangements produce an increase of the swelling,—and finally, that in one case, at all events, the thrilling tumour of the neck subsided under the use of iodine,—there is good reason why we should not draw too strongly the line of demarkation between the diseases.

With respect to the enlargement of the eye-balls, we may observe that it occurs last in the chain of phenomena, and probably arises from an augmentation of the vitreous and aqueous humours of the eye. Both eye-balls are simultaneously and equally affected, and so far from signs of sanguineous congestion existing, the eye has a singularly clear and transparent appearance, which in some cases amounts to a morbid brilliancy. There is a peculiar staring expression caused not only by the prominence of the ball, but from the line of the sclerotic coat which is seen surrounding the cornea to a greater or less extent. Under these circumstances a maniacal expression is produced. As the disease advances, the protuberance of the globe may become extraordinary. It protrudes outwards and downwards, and the lids, being no longer able to cover the eye, the patient sleeps with the eyes open; yet it is a most singular fact that the power of vision is not in any way injured, nor is the patient rendered liable to ophthalmia. I have known a case in which, for upwards of a year, the eye was never closed, yet in which no vascularity of the conjunctiva, nor any form of ophthalmia, ever occurred.

When emaciation takes place, the expression of the countenance produced by this staring, protuberant, and never-closing eye, is most painful and extraordinary. Yet so far as the eyes are concerned, the patients make little or no complaint. What they principally suffer from is the palpitation of the heart, the throbbing in the neck, and the sensation of fulness in the head and constriction when the head is bent forward so as to compress the thyroid tumour.

In most instances we observe a want of proportion between the force of the pulsations in the arteries of the neck and those in other parts of the system. The carotid and thyroid arteries may pulsate with vehemence, so as to give the idea that all the vessels of the neck are enlarged and in a state of morbid activity, yet the radial pulse be small and weak, and only rapid or irregular according to the state of the heart's action.

The exciting causes of this affection are various, but all seem to have acted first on the heart. Amenorrhœa, with or without hysteria, is a common cause. In young women, mental anxiety and the effect of terror may produce it. I have known a remarkable instance of the latter cause inducing the disease in a lady who had previously been healthy. In a case of the disease in the male subject, long continued hemorrhage from piles was assigned as the cause.

It has not been found associated with any form of carditis, or to be produced by hepatic disease. Indeed, all that we can say as to its nature amounts to this, that it is a special form of cardiac neurosis, which may eventuate in organic disease. Whether the nervous excitement is propagated to the arteries in the neck is a question I have often proposed to myself, for there is something in their action more than can be explained by the force of the heart. If we compare the pulsations of the carotid with those of the radial arteries, the difference is most striking; the former being violent in a high degree, while the latter are small and weak, only corresponding with those of the carotids in their frequency. Exceptional cases are, however, met with.

In this affection we commonly observe that double pulsation of the arteries to which we have before alluded; and its existence in the neck alone is another evidence of a local vascular excitement.

This pulsation is perceptible to the hand and ear, and has precisely the character of the double pulsation in an aneurism. It is, as it were, its diminutive. Yet this condition is peculiar to the carotid arteries, and its existence could never be conjectured from any character of the radial pulse.

The disease is capable, if not of cure, at least of great amelioration. The enlargement of the eye-balls diminishes, so that the staring expression disappears, though a certain fulness of the globe may remain. The thyroid gland is lessened in volume and appears to become more solid; it loses the violent pulsation and the purring thrill, or the latter may be confined merely to certain parts of its surface. The excitement of the arteries subsides, and the heart becomes comparatively tranquil, yet these changes require a long period for their completion. I am not in possession of any proof of the complete cure or resolution of the disease, when fully formed, though we have no reason to believe such an occurrence impossible.

Dr. Parry[1] has given several cases of enlargement of the thyroid gland in connexion with affections of the heart. In the first of these cases the patient had been attacked by acute rheumatic fever consequent on her lying-in. This was followed by palpitation of the heart, which gradually increased in force and frequency until Dr. Parry commenced his attendance, when it was so vehement that the whole thorax was shaken at each systole of the heart. The pulse was 156, very full and hard, irregular in strength, and intermitting at least once in every six beats. She suffered from symptoms resembling cardiac asthma, attended with slight hæmoptysis, and had also frequent and violent pains about the lower portion of the sternum. "About three months after lying-in," says Dr. Parry, "while she was suckling her child, a lump about the size of a walnut was perceived about the right side of the neck. This continued to enlarge till the period of my attendance, when it occupied both sides of the neck, so as to have reached an enormous size, projecting forwards before the margin of the lower jaw. The part swelled was the thyroid gland. The carotid arteries on each side were greatly distended; the eyes were protruded from their sockets, and the countenance exhibited an appearance of agitation and distress which I have rarely seen equalled. She suffered no pain in her head, but was frequently affected with giddiness."[1]

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[1] See "Collections from the unpublished Medical Writings of the late Caleb Hilliard Parry, M. D.," p. 111. London: 1825.

This patient soon afterwards died with the usual symptoms of anasarca and disease of the heart. No dissection is reported.

In the second case, a lady in consequence of a fright became subject to palpitation of the heart and various nervous affections, and in about a fortnight she observed a swelling of the thyroid gland, which subsequently varied at different times so as to be once or twice nearly gone. When seen by Dr. Parry, the gland was swelled on both sides, but especially on the right; the pulsation of the carotids was very strong and full, but predominating on the right side. She stated that she had formerly been subject to headachs, which had ceased since the commencement of the swelling. The pulse was 96, small, hard, and regular. Ten ounces of blood were taken from the arm, and this operation was followed by evident diminution of the tumour; and after her next menstrual period the swelling of the thyroid, which had returned, was found to have almost disappeared. On the next ensuing period, the tumefaction increased previous to the discharge, when it again diminished.

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[1] The combination of disease of the heart and enlargement of the thyroid gland is noticed by Flagini. See his "*Collezione d'osservazioni e riflessioni di Chirurgia,*" Roma, 1800, vol. iii. p. 270. A case is quoted in the Medico-Chirurgical Review, vol. i., from the New England Journal, October, 1820, in which, after violent palpitations, a pulsating tumour extended high above the right clavicle, and presented a strong thrilling sensation. The symptoms subsided soon after an attack of hæmatemesis.

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The next three cases are examples of the combination of enlargement of the thyroid with symptoms of organic disease of the heart. The tumefaction in all of them was attended with increased action of the carotids, and in two cases the thyroid swelling evidently followed a long-existing cardiac disease. In the sixth case, a married woman, with a very long neck, who never had had a family, after keeping her feet a quarter of an hour in cold water for the relief of chilblains, was attacked with violent pain in the region of the heart. For five years after that period these attacks used to return, and were subsequently attended with palpitations and attacks of difficulty of breathing, with globus hystericus. During the palpitation, and indeed at other times, she had violent beating in the head and pulsation of the neck; and after one of these attacks, which was unusually severe, the thyroid gland began to swell. The subsequent reports of this case furnish nothing of importance. Two more cases are given, but they do not contain any novel observation. No dissection is recorded by Dr. Parry, nor was the enlargement of the eyes observed but in a single case; and, indeed, if we except the second, and perhaps the sixth, case, they are examples either of enlargement of the thyroid gland succeeding to a long-existing organic disease of the heart, or of accidental disease of that organ in a case of bronchocele.

This disease, however, remained but little known until the publication of Dr. Graves's Lectures in 1835, afterwards embodied in his "Clinical Medicine." This author first pointed out the distinction between the enlargement of the thyroid in these cases and that in ordinary goitre. He suggested that the thyroid body might be slightly analogous to the tissues properly called erectile, and that the globus hystericus is not necessarily a simple nervous affection, but really arises from a temporary enlargement of the thyroid.

I communicated the following case of this affection to Dr. Graves after hearing his clinical lecture on the combination of palpitation of the heart with enlargement of the thyroid gland. A young lady, of delicate make and nervous constitution, became affected with various forms of hysterical and neuralgic disease. She complained of debility upon exertion, and lost flesh and colour; she suffered from palpitation of the heart; and in the course of a few months it was observed that the pulse was never under 120. It frequently rose to nearly 140, and was small and compressible. The contrast between the action of the radial and carotid arteries was most remarkable, the pulsations of the latter being violent and jerking, attended with a deep bellows murmur, and conveying the idea that the arteries themselves were enlarged. The action of the heart had that sudden, sharp, and jerking character which is found in nervous palpitations, while its rate never fell below 120. The eye-balls were now observed to enlarge gradually, until at length their condition imparted to the countenance an unearthly expression. The tumefaction continued to increase until the globes of the eyes appeared to protrude from the orbits, looking downwards and forwards, and exhibiting a zone of the white sclerotic round the entire of the cornea of at least two lines in breadth. The lids could only be half closed; and the appearance of this lady during sleep, with these great brilliant eyes yet open, can never be effaced from my memory. It was remarkable that the conjunctiva was never vascular, nor were any symptoms of ophthalmia developed, such as we see occurring in the open eye, which attends on the facial paralysis described by Sir Charles Bell. Notwithstanding the unnatural enlargement of the organs, there was no alteration in the power of vision. The thyroid gland was increased in volume, and formed an elastic tumour, in shape somewhat resembling a horse-shoe. It was at first soft, but soon became harder, though still elastic. It very soon attained its maximum development, forming a tumour about the size of a small orange, after which it did not continue to increase. The condition of the eyes was to a certain degree variable, but they remained greatly enlarged up to the period of death. She suffered little from the state of the heart, thyroid, or eyes, her principal distress being the occurrence of severe facial neuralgia. Little change occurred in the symptoms for upwards of two years, and this lady sank with general anasarca and pulmonary congestion; in short, with the symptoms generally attributed to dilatation of the heart.

CASE XXVI.—*Long-continued excitement of the Heart in a male subject; Enlargement of the Eye-balls and of the Thyroid Gland; Increased action of the Vessels of the Neck, with murmur in the tumour similar to that of Aneurismal Varix; Ultimate subsidence of the morbid action of the heart, with diminution and hardening of the Tumour in the Neck.*

John M'Keon, aged 48, tall, spare, and dark-complexioned, of a nervous and sensitive temperament, was admitted to the Meath Hospital during the year 1838, labouring under violent palpitation of the heart, general arterial excitement, and enlargement of the thyroid gland. His history was, that he had for many years been healthful, and of regular habits, never addicted to intemperance, and working laboriously at his occupation. About seven years ago, after a hard day's work and exposure to inclement weather, he was attacked with violent palpitations of the heart, unaccompanied by pain or any other symptom except slight vertigo. He attributed these to obstinate constipation, from which he had long suffered. They subsided after three months' continuance, and from that time he continued healthy, with the exception of a small tumour that appeared some years ago in the region of the thyroid, but which gave him no annoyance. In January, 1838, the palpitations again returned, and in about six weeks became so violent as to cause him to seek admission into hospital. At that time he was much emaciated, and suffered from general debility; it was chiefly the palpitation and arterial excitement of which he complained, the thyroid enlargement causing him little or no suffering. His appetite was good, and he slept well; pulse 96. His appearance, however, was very peculiar, and at once arrested attention. Situated over the trachea, and corresponding to the part occupied by the thyroid gland, was seated a large tumour, of soft and flabby consistence, most prominent laterally, and resembling in many respects a bronchocele of moderate growth. A remarkable thrill, resembling that perceived in aneurismal varix, was communicated to the hand placed on the tumour, particularly over its left lobe. Large and swollen veins ramified over its surface; and when the stethoscope was placed on it an intense musical murmur was audible. The same existed in the carotids, but the thrill in the tumour appeared to be independent of these vessels. His eye-balls were very prominent and enlarged; he had no stridor or dysphagia, but small portions of food occasionally pass into the nares. His heart pulsated violently between the fourth and fifth ribs, but no murmur accompanied its sounds. The carotids pulsated visibly, as also the left subclavian. A particular examination of the tumour discovered that when the finger and thumb were made to grasp and compress the vessels of the neck, the vibration at first became stronger, but as the pressure was increased, it altogether subsided, though the impulse of the vessels continued. The stethoscope being applied over the tumour pending the pressure on the left side, the murmur ceased on the corresponding side, but the thrill, and consequently the sound, continued on the opposite side. The tumour, which at the time of his admission into hospital measured 16½ inches round the most prominent part to the sixth cervical vertebra, after a short period was reduced to 15. The palpitations had become less violent, the thrill and the tumour, particularly on the right side, greatly decreased, and he left the hospital greatly improved in every respect.

In a short time after, however, all his former symptoms returned in a more aggravated degree, and he also suffered from diarrhœa and hæmorrhoids. He was again admitted into hospital. The palpitations had returned, and he complained of violent pulsation of the abdominal aorta. There was no material change in the tumour from that before described, except that it was somewhat diminished in size, but the thrill and musical murmur continued as intense as ever. The vessels of the neck were now enormously swollen, yet no immediate contact could be discovered to exist between the carotids and the tumour. His heart pulsated violently between the sixth and seventh left ribs. An occasional intermission existed in the beats of the heart, of which the patient himself was conscious. The first sound was remarkably loud, the second, shorter, sharper, and weaker than natural; a kind of muscular murmur accompanied the heart's action, which was apparently created by the violence of the impulse, but no valvular murmur accompanied either sounds. The pulsation of the abdominal aorta was excessively violent, and the slightest pressure over it created a murmur. His eye-balls still continued prominent and enlarged. There seemed in this man to be a tendency to irregular distribution and division of the arteries. The ulnar artery came off much higher up than usual, and ran for some distance superficial. The radial sent off a large branch which crossed the arm above the wrist. His treatment consisted in the use of anti-nervous medicines, anodynes at night, and subsequently the long-continued use of digitalis. Under this treatment he regained flesh and strength. The arterial excitement became very much reduced, and the pulse seldom rose higher than 80. The intermission in the beats of his heart disappeared after a fortnight, and the pulsation of that organ became much reduced in force. He left the hospital after six weeks, and since that time has continued to improve, as the following observation, made several years since his last leaving the Meath Hospital, will show:—Action of the heart tranquil, and no murmur accompanied the sounds. The enlargement of the eyes had almost entirely subsided, and the tumour itself was much reduced in volume, and to all appearance solid. There was no thrilling murmur in any part of it save in the course of a tortuous vein which ran from above downwards over its anterior surface. This vein had a loud murmur and a slight purring thrill.

For some years subsequently I had opportunities of seeing this patient, and of observing the decline of the symptoms. It appeared to me that the process of improvement began in the heart, the action of which gradually became more natural. In the eyes improvement was next exhibited, yet even after the enlargement had subsided, they preserved a certain intensity of expression. It is difficult to say how much of this was natural, for the patient was a man of erect carriage, and of a bold and determined character. On my last examination I found that all signs and symptoms of cardiac affection had subsided; the thyroid tumour had become everywhere solid; it was nearly hemispherical; it had lost its pulsation, and no trace of thrill or of the humming sound could be discovered. The vein formerly observed was as large as the finger, and traversed the tumour a little to the left of the mesian line, lying in a sulcus formed in the substance of the tumour, above the edges of which it was slightly raised. The coats of this vein, throughout the entire course of which a purring thrill and a singularly hoarse murmur were perceptible, appeared extremely thick. The throbbing of the carotids was gone, and the jugular veins were in a perfectly natural state.

There are some circumstances in this case which are worthy of special notice. The first attack of disease occurred seven years before the patient's admission into hospital, when he had violent palpitation of the heart, which, after continuing for three months, subsided. He remained healthy until about ten months before his admission, with the exception of a small tumour of the thyroid gland, which gave him no annoyance, although it had existed for several years. This seems to show that in this special combination the thyroid gland may remain for a time indolent, and again take on a morbid action when the heart becomes a second time affected.

Lastly, it is worthy of notice, that the arterial excitement in this case was not confined to the vessels in the neck. The patient, at a time when the disease was at its height, suffered from increased action of the abdominal aorta. On the other hand, it is certainly true, that in many cases an extraordinary disproportion may be found between the force of the arteries in the neck and in the extremities.

It is probable that the following classification of these cases may be adopted:—

1. The pulsating and thrilling thyroid tumour succeeding to an increased action of the heart.

2. An indolent and non-pulsating tumour existing for a length of time without any remarkable alteration in the action of the heart, but, consequent on the attack of palpitation, taking on the character of aneurismal varix.

It may be that in this last division of cases there is a greater liability to the production of large tumours of the neck, which consist, on the one hand, of the altered thyroid tumour, and on the other, of vast dilatations of the veins, forming, as it were, separate tumours on each side of the gland. A case of this sort is noticed by Sir Henry Marsh, and there is a cast of a great tumour, exhibiting prodigious dilatation of the veins, as well as of the thyroid, in the Museum of the Richmond Hospital. In this case pulsation and fremitus existed to a remarkable degree.

The following example of this condition was communicated to the Pathological Society of Dublin in 1841, by Sir Henry Marsh. The patient was a person of tall stature; she suffered from palpitation of the heart and dyspnœa, increased by exercise or mental emotion. The action of the heart was irregular and peculiar: three beats followed in succession; the first, strong and distinct; the second, closely following, had a double character; and the third appeared more distant. The interval of repose then succeeded. There was no bellows murmur. The patient presented all the characters of the disease under consideration, namely, remarkable engorgement of the veins of the neck, prominence and protrusion of the eye-balls, and enlargement of the thyroid gland. It was observed that the swelling of this organ increased or diminished according as the action of the heart was more or less violent, and this swelling was attended with corresponding tumefaction of the veins of the neck, so that the external jugular veins formed tumours on each side, giving an extraordinary appearance to the patient. The prominence of the eye-balls, however, was not so well marked in this case as in others which he had seen. After a long illness, death occurred from gangrene of the extremities, which had been preceded by erysipelas and anasarca.

On dissection, the thyroid gland exhibited an irregularly lobulated surface, the lobules or cysts containing a quantity of clear fluid. The internal jugular vein on the right side was much dilated, measuring when emptied by puncture an inch and a half across. It was filled with dark fluid blood. One of the enlarged lobes of the thyroid body lay over the carotid artery. The lungs were forced upwards.

Both auricles, but particularly the left, were found much dilated. The left ventricle was dilated and hypertrophied, though not to a very great degree. The auriculo-ventricular valves, on both sides, exhibited thickened margins; the disease apparently proceeding from depositions of fatty granular matter under the membrane. The right valves were more affected than the left.

The following case was communicated to me by Professor Smith, and is of value, as showing that the thyroid arteries are engaged in the disease.

An unmarried woman, of florid complexion, and with every appearance of robust health, was admitted into the Richmond Hospital, under the care of the late Dr. M'Dowel, complaining of palpitation of the heart and occasional vertigo. She exhibited the physical signs of hypertrophy of the left ventricle, but without any decided evidence of valvular disease. There was a considerable enlargement of the thyroid gland, principally owing to hypertrophy of its right lobe. The thyroid arteries could be felt pulsating strongly. The eyes were large and brilliant, but were not protruded. Shortly after her admission she was seized with apoplexy, which speedily proved fatal.

On examination the left ventricle was found hypertrophied to a great degree, and its cavity much dilated,—the slightest appearance of the first stage of disease of the aortic valves existed,—but they were still competent to close the orifice. The thyroid arteries were greatly enlarged and remarkably tortuous. The brain exhibited the usual appearances of apoplexy with extravasation. The thyroid gland was enlarged, but no dilated veins could be seen ramifying on the surface of the neck.

The last case of this affection which I have observed is that of a gentleman, aged 33, who had suffered from two attacks of the swelling in the neck. The first appearance of the disease took place about four years ago, when he found that his neck was gradually enlarging. This patient was of a nervous temperament, and had been exposed to much mental annoyance. His health, too, had suffered from intense application to study. Under the influence of change of air and occupation the swelling of the neck gradually disappeared, and in about a year subsided. Thus he continued until six months since, when, after severe mental exertion both by day and night, the symptoms returned, and he began to suffer from difficulty of breathing, and a feeling of constriction in the neck. The circumference of the neck progressively increased, so that he was obliged to enlarge his shirt-collars again and again. His eyes were suffused and red, but he did not suffer from palpitation of the heart nor from dysphagia. At this time, in consequence of being informed by his medical attendants that his disease was aneurism, a great increase of nervous excitement, and consequently of the local disease, took place. When I saw him, the circumference of the neck was greatly augumented, giving something of the tippet-like appearance which is occasionally observed in aneurism of the aorta; but this was not produced by œdema. The thyroid gland was greatly enlarged, forming a flat tumour, on each side of which vast dilatations of the veins, forming elastic swellings having a sacculated appearance, could be seen. I could not find any fremitus, but the central portion of the tumour had a diastolic pulsation, which anticipated the pulse of the radial artery by a short but distinct interval. A deep systolic murmur, loudest at the top of the sternum, and feebly heard under the clavicles, was perceptible. The heart's action was excited, but regular, and no valvular murmur could be found.

Although differing in some particulars from those already given, this case is worthy of study. The eyes were to a certain degree enlarged, and the original cause of the disease was not alone nervous excitement, for at the time of the first attack the patient was living in a district where goitre is endemic. The important points of difference were the existence of the venous tumours, and the absence of the thrill and sounds which so closely resemble those of aneurismal varix. The seat of the systolic murmur was in all probability in the thyroid arteries.

If we now review what has been said on this disease, we must admit that our knowledge of the affection is still very imperfect, for although cases of this description are not unfrequent, yet we possess little information derived from dissection as to their pathological nature. A few carefully conducted examinations of the state of the heart, aorta, carotid and thyroid arteries, and, lastly, of the eyeballs and venous system of the neck, would supply an important deficiency in cardiac pathology. It is true, that we might thus learn the result, rather than the cause of the disease, but even this would be of great service to practical medicine.

In the present state of our knowledge we may conclude that this disease, so well marked in its triple character, is originally a functional and not an organic affection; for, although in the cases observed by Sir Henry Marsh and Professor Smith, organic alterations of the heart were found, yet there is good reason to believe that these changes were long preceded by a special nervous excitement. We find that the confirmed disease is capable of resolution, as in Case XXVI. We find also, that minor forms are susceptible of cure, and that whether we consider the subjects most liable to the affection, or its ordinary exciting causes, there are strong reasons for holding that the disease is originally a neurosis of the heart, and, perhaps, also of the cervical vessels themselves. To these considerations may be added the important fact, that in hypertrophy of the heart, as it is commonly met with,—in other words, in that condition which appears most favourable for causing an increased flow of blood to the neck and head, this combination is rarely to be seen.

The affection of the thyroid gland itself differs from that in ordinary bronchocele in several points of view. In most cases, as Dr. Graves has observed, it becomes stationary after having attained a certain development, and though a greater amount of deformity may occur than that which he thinks possible, yet I have never seen in this disease the vast enlargement of the thyroid which occurs in ordinary bronchocele.

But the important distinctive mark is the existence of the peculiar thrill similar to that of aneurismal varix. In some patients this sign is equally developed over the whole surface of the tumour, while in others it is more localized. We cannot as yet declare whether the seat of this thrill and extraordinary murmur is in the branches of the thyroid artery or in the veins, or whether there may not be a morbid condition of the entire capillary system of the tumour. Finally, we observe venous murmur in the superficial veins, and a bellows murmur, the seat of which is probably in the thyroid arteries, but which may occasionally be heard at the upper portion of the sternum and under the clavicles. This murmur occurs without any disease of the aorta or of the heart.

In cases of recovery, the thyroid tumour becomes smaller and apparently solid, and the thrill and murmur both disappear, although they may remain in some of the altered veins of the neck.

The principal supply of blood to the thrilling tumour appears to be from the inferior thyroid arteries. In Professor Smith's case the carotids and superior thyroids were unaffected, while the inferior thyroids were so enlarged as to equal the brachial artery in dimensions. The fact, as observed in Case XXVI. of our being able to command not only the diastolic pulsation, but the purring thrill, by pressure exercised at the base of the tumour and immediately above the clavicles, confirms this opinion; and it will be recollected that while the thrill ceased, the impulse of the carotids continued,—it will be recollected also, that the first effect of pressure was to increase the intensity of the thrill and the loudness of the sound, but that both thrill and sound disappeared when the compression was carried still further. So far as a single observation can go, this indicates that the principal seat, both of the thrill and murmur, is in the dilated veins, and we may hold that the augmentation of these signs took place in consequence of the increased flaccidity of the vessels, when pressure caused a diminished supply. We here apply the principles indicated by Dr. Corrigan, when he speaks of murmur and tremor in connexion with disease of the arteries.[1]

Our knowledge of this disease from dissection is so scanty that little that is satisfactory can be said with respect to the state of the thyroid arteries and of the veins. In the case observed by Professor Smith, the thyroid arteries were enormously enlarged and tortuous, and in that by Sir Henry Marsh, the right internal jugular vein was found distended. Indeed, there can be little doubt that a disposition to dilatation of all the veins of the neck exists in this disease.[2]

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[1] On Permanent Patency of the Mouth of the Aorta. Edinburgh Medical and Surgical Journal, vol. xxxvii. p. 230.

[2] In speaking of vascular bronchocele, Hasse observes that all the blood-vessels are amplified, the veins in particular forming very dense, capacious, often knotted plexuses, and the whole texture consisting apparently of a dense coat of vessels. The substance of the gland has almost entirely lost its granular character; it is flabby and dark red. After death the tumour collapses considerably, and can only be restored to its original size by artificial injection. The walls of the arteries and veins are attenuated; the dilated membranes of the vessels contain considerable clots, and capacious cavities are found filled with black coagulated blood, (see Dr. Swaine's translation, p. 387.) It is probable that there are more forms than one of the vascular bronchocele; and though we might thus designate the condition of the thyroid in the affection described in the text as a form of vascular bronchocele, it is obvious that the disease is but a link in a chain of various morbid actions.

The condition of the eye-balls is one on which dissection has not as yet furnished us with any information; but we cannot come to any other conclusion than that it is an example of double hydrophthalmia, or at least of an enlargement of the eye-ball itself. When we consider the nature of the vascular apparatus of the eye, and recollect also that in this disease the powers of vision remain unimpaired, and that the eye shows no symptom of increased vascularity, we cannot but conclude that the enlargement is owing to an actual increase in the vitreous and aqueous humours of the eye. There is not the slightest evidence that the protrusion of the eye-balls is symptomatic of disease of the brain.[1] Nor can we admit that the protrusion arises from œdema of the orbital cellular structure, for it is a remarkable fact, that in some cases of the greatest emaciation, this singular condition becomes most prominent. This was remarkably seen in the case which I communicated to Dr. Graves.

The enlargement of the eyes may appear in a sudden manner. Thus in a case observed by Dr. Adams, the appearance of the eyes presented nothing remarkable until after a long-continued fit of coughing and retching. On the following day the symptom attracted the attention of the lady's friends.

We may expect that some light will be thrown upon the functions of the thyroid gland by the study of this disease. In speaking of the connexion between bronchocele and affections of the head, Dr. Parry suggests that the thyroid gland "is intended in part to serve as a diverticulum in order to avert from the brain a part of the blood which, urged with too great force by various causes, might disorder or destroy the functions of that important organ." The fact, long known, of the connexion between the thyroid gland and the state of the uterine function, should be considered, and it is to be remembered that in cases of the combination now before us, Dr. Parry observed not only the increase of the tumour immediately before, but its subsidence after the menstrual discharge; and he has further shown that venesection had the effect of reducing the swelling of the neck. Finally, some analogy may be found between this condition of the thyroid gland and that of the liver, in cases of obstruction or weakness of the right side of the heart.

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[1] The fact of the simultaneous enlargement of both eyes and the absence of symptoms of abscess of any portion of the brain, to say nothing of the general history of these cases, is quite conclusive.

The following conclusions appear justifiable in regard to this peculiar and still obscure affection:—

1. That under certain circumstances the action of the heart may become permanently excited, as shown by its rapidity, irregularity of action, and increased force; and that this state is attended with three remarkable epiphenomena, namely, the turgescence of the thyroid gland, the increased action of the arteries of the neck, and the enlargement of the eyeballs.

2. That this condition is not attended with fever, or signs or symptoms of cardiac inflammation, but is more related to functional disturbance.

3. That the disease is most commonly observed in the female, associated with hysteria, neuralgia, or uterine disturbance; but that it may occur with all its characteristic phenomena in the male.

4. That this combination of diseased actions may occur at a great variety of ages, from puberty upwards.

5. That it exhibits exacerbations and remissions at various periods, which appear to depend on the condition of the heart's action.

6. That the enlargement of the thyroid gland arises quite independently of the ordinary exciting causes of endemic bronchocele.

7. That this enlargement is attended with a diastolic pulsation of the tumour.

8. That in addition to the diastolic throb, there are presented the usual physical signs of aneurismal varix in the gland.

9. That the existence of these signs, namely, the purring thrill and murmur, may be general or partial, and also vary in intensity in different parts of the tumour, and at different periods of the disease.

10. That in the more advanced periods these signs subside with the increasing solidity of the gland.

11. That various venous murmurs may exist in the jugulars, or in the large veins traversing the tumour, during the progress of the disease, and even after it has lasted for years.

12. That there is some probability that the sensation termed the "*Globus Hystericus*" may proceed from the temporary existence of the first stages of this affection.

13. That the increased pulsation of the arteries of the neck cannot be explained by cardiac regurgitation, or by any determination of blood to the brain; nor is it any evidence of general arterial excitement.

14. That under these circumstances the double sound and impulse are often developed in the carotids.

15. That the enlargement of the eye-balls is not necessarily attended with any alteration of vision, nor does it appear to predispose the eye to inflammatory disease either of its external covering or internal structures.

16. That this enlargement is variable in amount during the progress of the case, and that it may subside to a great degree, if not altogether.

17. That in fatal cases of this affection the morbid conditions which have been observed are dilatation and hypertrophy of the heart, enlargement of the inferior thyroid arteries, and dilatation of the jugular veins.

18. That a mixed case of the disease may be met with, in which a previously existing bronchocele of the ordinary kind is influenced by the occurrence of nervous or organic disease of the heart.

19. That the essence of the disease appears to consist in functional disturbance of the heart, which may be followed by organic change.

CHAPTER IV.

WEAKNESS OR DEFICIENT MUSCULAR POWER OF THE HEART.

As a weakened state of the heart, no matter from what it may arise, indicates a certain course to the physician, we shall, even at the risk of repetition, trace out its most ordinary causes. They are as follows:—

Weakness of the heart in connexion with muscular atrophy or emaciation—occurring in the combination of cardiac dilatation with hepatic and pulmonary disease—as a result in cases of pericarditis or endo-pericarditis—in connexion with fatty degeneration of the organ—in fever, independent of softening of the heart—and, lastly, in connexion with the softening of the organ as a result of essential fevers, and especially typhus.

We thus see that the weakened condition of the heart may be presented under various circumstances. Its principal causes, however, are reducible to two great heads, namely, weakness from diminished innervation, independent of organic disease, and again, that debility which is consequent on or associated with organic change of some kind.

The condition of ordinary syncope is referrible to the first of these heads; the completeness of the syncope being in proportion to the amount of temporary paralysis of the heart. In this condition the loss of contractile power is temporary, but it is probable that in some diseases a more enduring state of debility of the heart occurs, yet quite independent of anatomical change in the organ.

It is still to be determined whether the heart, or any one of its cavities, is liable to paralysis resulting from a primary lesion of the nervous centres or the ganglionic system; or again, whether obstruction of the coronary arteries can cause a semi-paralyzed condition, analogous to that which has been already described as occurring in the extremities from chronic arteritis.

Atrophy of the Heart.—Of atrophy of the heart, considered as an idiopathic affection, we know little or nothing, nor are we able to specify any symptom of this condition in cases of general emaciation; for in chronic tuberculosis of the lung with atrophy of the heart, no peculiarity of the circulation has been observed. It is in phthisis that we most often meet with extreme atrophy of the heart, a condition to be explained by referring to the diminished amount of circulating fluid on the one hand, and the process of absorption of the red tissues on the other. In this way not only the voluntary but the involuntary muscles are diminished in volume and power, and become pale and flabby, as is exemplified not only in the heart, but in portions of the digestive and generative systems. To such a degree is this process carried in the stomach, that the organ may resemble a membranous bag of extreme tenuity, as in the *ramollissement avec amincissement* of Louis. Other examples may be given, among which should be noticed the atrophy of the uterus, under the same general condition, as first described by Professor Montgomery.[1] In the heart this process of atrophy is not confined to the muscular structures alone. The valves may be singularly atrophied and cribriform, as described by Mr. King, of London, and also by Dr. Adams and Professor Smith. In a patient who died of phthisis at a very advanced age, and in whom the aortic valves were the seat of the alteration, I found the filaments corresponding to the perforations to be as delicate as a spider's thread.

Dr. Hope states, that in atrophy without any other change in the organ, the heart generally contracts on itself so as to diminish its cavities. It is under these circumstances that the heart of an adult resembles that of a child, a condition described by many authors, and by no means uncommon. But when in phthisis the fatty degeneration engages the organ, its volume may be even larger than natural.

I have seen no example of the production of this atrophy by excessive bleeding, as mentioned by Laennec, or from the pressure of false membranes, of which a case is given by Bouillaud.

Weakness of the Heart in Pericarditis—To every one who has seen fatal cases of this disease, the condition of the patient for some time before death appears to indicate that the left ventricle has lost much of its contractile force. The smallness, irregularity, and feebleness of the pulse, the coldness of the surface, the pallor of the countenance, and the frequent tendency to faint, all indicate extreme weakness of the systemic side of the heart. The symptoms in question are supposed by many to arise from the pressure of the effused fluid, an explanation difficult to be received when we recollect how little the function of the heart is disturbed in its dislocations from eccentric pressure. It is more than probable that a condition of the ventricular muscles analogous to that of the intercostals in advanced cases of empyema does really occur; and we may draw a close analogy between the yielding state of the intercostal muscles in pleurisy and the debility of the cardiac muscles in pericarditis. In both we have the common condition of inflammation of a tissue in close connexion with the muscle itself, and in both we observe, first, the evidences of excitement, and next, those of depressed vitality of the contiguous muscles. Whether it be that in these cases inflammation of muscular tissue itself is associated with that of the serous membrane, is yet to be determined; but that such is the fact appears not improbable, particularly as myocarditis has been observed in certain cases of pericarditis.

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[1] Dublin Journal of Medical Science, First Series, vol. xxvii. p. 161.

It has been already shown that the non-recognition of this cause of debility of the heart has led to grave errors in the treatment of pericardial inflammation, and that the life of the patient is often sacrificed by perseverance in an antiphlogistic regimen at a time when the heart was losing its contractile force, and when stimulation had become necessary. We are still in ignorance of any direct means by which the first stages of this important change may be indicated, but the progress of clinical observation will throw great light on this subject.

It is unlikely that under these circumstances the debility of the heart can be combated by stimulants with the same success as in the non-inflammatory softening or the debility in typhus, but its occurrence should make us suspend antiphlogistic treatment, and theoretically it indicates stimulation.

According to Hasse, we may assume that no one form of carditis can occur in a high degree without implicating more or less all the textures of the heart. "We must, however," he says, "guard against confounding with carditis those cases of pericarditis, and of pleurisy of the left side, in which the substance of the heart is either flaccid, pale, and softened, or here and there dark-coloured and pulpy. As well might a diaphragm, softened and discoloured by inflammation of the superincumbent pleura, be reckoned as inflamed. In both instances the influence of the neighbouring inflammatory process is too obvious to be called into question."[1]

This author has admitted the dynamic origin of the dilatation of the intercostals and diaphragm in pleurisy, which I long since demonstrated, and analogy leads us to apply the same reasoning in the case of pericarditis. It is still to be determined how much of mere paralysis, and how much of an actual inflammatory state of the cardiac muscles, concur in the production of the weakened or flaccid condition.

Weakness of the Heart from Dilatation associated with pulmonary and Hepatic Disease.—It is only necessary to notice this form here. Its history has been already given in the preceding pages.

We shall now proceed, in a separate chapter, to examine those cases of weakness of the heart which proceed from the fatty degeneration of the organ; after which, that important form of weakness of the heart so commonly met with as a secondary disease in the essential fevers of this country may be studied with advantage. This inquiry, however, refers to a special condition, and we shall not enter upon it until the general treatment of the organic diseases of the heart has been discussed.

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[1] Hasse, Pathological Anatomy, Swaine's Translation, p. 201.

CHAPTER V.

ON FATTY DEGENERATION OF THE HEART.

THE accumulation of fat upon the surface or in the substance of the heart has long been recognised. But it is only within our own time that the subject was properly examined. Laennec, who has been followed by Hope and by Hasse, describes two forms of the affection; and though his account of the disease is but meager, the researches of subsequent observers, including Rokitansky, have not established any case that may not be referred to either of his varieties.

In one form, fat is deposited more or less abundantly in the subserous cellular membrane, so as to produce a layer of fat enveloping the heart. Its thickness is variable, and its distribution generally most abundant over the right cavities. The muscular structure is as it were pushed before it, and commonly found pale, softened, and wasted. According to Hasse, the fat globules collect not only within the compartments of the subserous cellular tissue, but are freely deposited within the muscular substance, and even between its primitive fibres.

The colour of this deposit is generally yellow; and in extreme cases, on the sternum being raised, the heart appears as if it were jaundiced.

In the second variety, the adipose degeneration is supposed to commence in the muscular structure, and to be a true transformation. Rokitansky states, that in this condition the fat does not accumulate in masses, there being no fat globules included within fasciculi of cellular tissue, but it is beaded in minute microscopic granules, closely interlaced and imbedded among the primitive fibres of the heart's muscles. These have lost their transverse striæ; the fibrils are friable and easily reduced to minute molecules.[1] This condition affects the left, perhaps, more than the right ventricle, and is generally thought to be the most common cause of spontaneous rupture of the heart.

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[1] Hasse, Pathological Anatomy, Dr. Swaine's Translation, p. 170.

Can we in the present state of our knowledge declare that a strong line of distinction exists between these forms? I apprehend not, and think that at least the first form may induce the second. With both these forms, such as Rokitansky has described, the pathologists of Dublin have long been familiar, and excellent specimens of the conditions in question exist in our museums. In the collection at the Park-street School there was a remarkable example of rupture of the left ventricle; the entire heart being so changed that it was barely possible to trace any muscular fibre in it. The form of the organ remained unaltered, but so great was the disappearance of the muscle that it is difficult to understand how circulation could have been carried on.

Rokitansky is of opinion that this change occurs in hypertrophied hearts, which exhibit the signs of a former endocarditis and carditis. How far the previous existence of inflammation may have predisposed to the affection is still to be determined; but that the affection may occur without evidence of valvular or pericardial disease seems established by the case published by Dr. Cheyne.

If, however, we look on this matter in a practical point of view, we find that the general symptoms and history in both forms of the affection are much the same. It is very probable, however, that the chance of rupture of the heart is greater in the second than in the first form.

Let us now examine some of the cases which have been observed in Dublin.

CASE XXVII.—*Fatty Degeneration of both Ventricles, with steatomatous and earthy Deposits in the Aorta; Pulse irregular and intermittent; Death by Apoplexy.*

This case is given by Dr. Cheyne:[1]—A gentleman, aged 60, who had lived a sedentary life and indulged freely at the table, became subject to gout in the feet. His regular gout subsided, and he suffered from œdema of the ankles in the evening, for two or three years before his death. His pulse was occasionally intermitting. On the 3rd of February, 1816, he returned home much exhausted by a long walk, and suffering from a fluttering or palpitation of the heart. This was relieved by wine. In the evening he was attacked by a severe fit of coughing, and fell insensible. No paralysis followed this attack, but the patient was pale and confused. The pulse was extremely irregular and unequal. Bleeding and purgatives were freely used, followed by mercurials and diuretics, as the secretion of the kidneys was scanty. The lungs, however, became more loaded, and anasarca rapidly increased. On the 10th of April he was found in bed flushed, speechless, and hemiplegic. The paralysis remained up to the period of his death.

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[1] Dublin Hospital Reports, vol. ii. p. 217.

The only peculiarity in the last period of his illness, which lasted only eight or nine days, was in a state of the respiration. For several days his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration.

On dissection the brain presented nothing very remarkable, except an increased vascularity of the pia mater, particularly over the middle and posterior lobes of the cerebrum. The ventricles contained three or four ounces of fluid. The pericardium contained two ounces of fluid, and the heart was three times its natural size. The lower part of the right ventricle was converted into a soft, fatty substance; the upper part was remarkably thin, and it gradually degenerated into this soft, fatty condition. The cavity of the left ventricle was greatly enlarged, and its whole substance, with the exception of the internal reticulated structure and carneæ columnæ, was converted into fat. The valves were sound, and the aorta studded with steatomatous and earthy concretions.

This case is full of instruction. We see in a patient of sedentary and luxurious habits the development of the gouty condition, and consequent on this the establishment of a weakened heart, as shown by the irregularity of the pulse and the tendency to œdema.

After an over-exertion the fluttering sensation about the heart is suddenly increased, and at the time relieved by the use of wine, and this is followed by an apoplectic seizure, from *which recovery takes place without any paralysis.* The patient is largely depleted, debarred from his usual stimuli, and dropsy sets in, with increased irregularity of the pulse. Another apoplectic attack, this time followed by hemiplegia, occurs, and the patient sinks, after exhibiting a peculiar character of respiration, doubtless a symptom of this condition of the heart. Dissection shows nothing but congestion of the brain, and a nearly complete degeneration of the heart into fat.

That a different line of treatment, at least with reference to the detraction of blood and the withdrawal of stimuli, would have been adopted by Dr. Cheyne if this case had occurred some years later, no one can doubt. Owing to the observations of Dr. Adams, the weakened state of the heart is now considered as the cause of the apoplectic seizures, and hence physicians are more cautious in reducing the system.

The next case is abridged from Dr. Adams's Memoir, published in 1827.[1] It is one of great interest, being in truth, the key to our knowledge of the subject, and having the same relation to the diagnosis of fatty heart that the case of aneurism by Dr. Beatty bears to that of aneurism of the abdominal aorta.[2]

CASE XXVIII.—*Repeated Apoplectic Attacks during a long series of years; Absence of Paralysis; Remarkable slowness of Pulse; Fatty degeneration of both Ventricles, especially the right.*

"An officer in the revenue, aged 68 years, of a full habit of body, had for a long time been incapable of any exertion, as he was subject to oppression of his breathing and continued cough. In May, 1819, in conjunction with his ordinary medical attendant, Mr. Duggan, I saw this gentleman: he was just then recovering from the effects of an apoplectic attack which had suddenly seized him three days before. He was well enough to be about his house, and even to go out. But he was oppressed by stupor, having a constant disposition to sleep, and still a very troublesome cough. What most attracted my attention was the irregularity of his breathing, and the remarkable slowness of the pulse, which generally ranged at the rate of 30 in a minute. Mr. Duggan informed me that he had been in almost continual attendance on this gentleman for the last seven years, and that during that period he had seen him, he is quite certain, in not less than twenty apoplectic attacks. Before each of them he was observed, for a day or two heavy and lethargic, with loss of memory. He would then fall down in a state of complete insensibility, and was on several occasions hurt by the fall. When they attacked him his pulse would become even slower than usual, his breathing loudly stertorous. He was bled without loss of time, and the most active purgative medicines were exhibited. As a preventive measure, a large issue was inserted in the neck, and a spare regimen was directed for him. He recovered from these attacks without any paralysis. Œdema of the feet and ankles came on early in December; his cough became more urgent, and his breathing more oppressed; his faculties, too, became weaker.

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[1] Dublin Hospital Reports, vol. iv.

[2] Ibid. vol. v.

"November 4th, 1819, he was suddenly seized with an apoplectic attack, which in two hours carried him off, before the arrival of his medical attendant.

"*Dissection* 56 *hours after death.*—The dura mater presented a natural appearance. The arachnoid membrane was separated from the pia mater by a fluid of gelatinous appearance. The substance of the brain was watery and of a yellowish white colour. There was some water in the ventricles. These cavities did not appear enlarged, but the foramen of communication between them was dilated. The coats of the carotid and middle arteries of the dura mater were quite white and opaque from bony deposition, but were pervious.

"The right lung was sound; the left was compressed, and adhered to the side of the thorax: about a pint of serum and quantities of soft fat, of a very deep yellow colour, filled up the space between the anterior mediastinum and the compressed lung, which was impervious to air, and must have been totally useless.

"The right auricle of the heart was much dilated. The right ventricle externally presented no appearance whatever of muscular fibres; it seemed composed of fat through almost its whole substance, of the same deep yellow colour as that which occupied the place of the left lung. The reticulated lining of the ventricle, which here and there allowed the fat to appear between its fibres, alone presented any appearance of muscular structure.

"The left ventricle was very thin, and its whole surface was covered with a layer of fat. Beneath this the muscular structure was not a line in thickness; it had degenerated from its natural state; was soft, and easily torn, and a section of it exhibited more the appearance of liver than of a heart. The septum of the ventricles presented the same appearance. In both ventricles, even in the lining fibres, yellow spots, where fat had occupied the place of muscular structure, were to be observed. The whole organ was remarkably light; the valves were all sound, except those of the aorta, which were studded with specks of bone, but elsewhere were cartilaginous and elastic, from which they derived a disposition to remain closed; a fluid gently injected from the ventricle would pass them; still when the heart was reversed and water poured from the ventricle upon them, their valves retained it; its weight was not sufficient to separate the edges of the thickened valves. There was much fluid blood contained in the heart.

"The liver was natural: the vena porta was unusually distended. The spleen was healthy in its structure, although enlarged; the other viscera presented nothing unusual."

In the memoir from which the above is extracted is given the particulars of another case, which, so far as I am aware, is without parallel in the records of medicine. It is that of a physician who during the last ten years of his life had suffered from repeated and sudden attacks of syncope, which, however, differed from ordinary fainting in the circumstances that the attack came on in a most sudden and unexpected manner, and in the same way went off, leaving no unpleasant effect. His age was 68, when he was suddenly attacked with symptoms resembling those of angina pectoris. He had severe pain in the chest, extending down the right arm, and attended with numbness. There was dimness of vision and rapid vertigo, but he did not faint. From that moment his breathing became oppressed, *and he discovered that the pulse, which was unaccountably weak in the left arm, had altogether disappeared from the right.*

This patient lived for six weeks, suffering from difficult respiration and declining strength, yet during the whole of this time *the most careful examination failed to discover any pulse in any artery of the body.* The action of the heart was not sensible to the hand, and on the application of the ear an obscure undulating sensation was all that could be observed. Dissection showed some recent pleuritis of the right side; the lungs were healthy; the heart was large, flabby, and of a yellow colour, from fatty deposition. All the cavities were distended with fluid blood; the semilunar valves and the aorta were completely ossified; but the bony or earthy deposition was not confined to the aorta; it extended to the coronary arteries, which were so completely converted into bone as to be quite solid, having no perceptible cavity except at the distance of an inch from their origin.

In explaining the extraordinary phenomena in this case, Mr. Adams leans strongly to the opinion that they are to be attributed to a more or less paralyzed state of the heart, resulting from the obstruction of the coronary vessels. The suddenness of the failure of the pulse is most remarkable; and that it was connected more with the weakness of the heart than the condition of the aortic valves is obvious, when we consider the symptoms of valvular obstruction, and the fact that the sounds of the heart were so singularly diminished, that to many it was doubtful whether its action could be discovered.

I have introduced this case here because that, with all its peculiarities, it comes into the category of weak hearts connected with fatty degeneration. It will be recollected that the heart was yellow from fatty depositions, and we shall presently see that an atheromatous or bony deposit in the aorta is a not infrequent complication of this affection. The same condition, extended to the coronary arteries, may have been the last step in the morbid processes in this case, and a semi-paralyzed state of the already weakened heart the immediate result.

A comparison of this case with that which Dr. Graves and I have given, of paralysis of the right lower extremity resulting from arterial obstruction, which apparently commenced in portions of the vessel not the farthest removed from the centre,[1] strongly corroborates the views that Dr. Adams has taken of the cause of failure of the heart's action, which in all probability was the more easily induced by the previously weakened state of the organ.

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[1] Dublin Hospital Reports, vol. v. Report of the Meath Hospital.

The next observer of this disease is Professor Smith, who has enriched our knowledge of the subject by his discovery of free oil in the blood, and by his observations on the production of air in the heart and veins after death.[1]

This author details the appearances on dissection in two cases. In one the immediate cause of death was rupture of the left ventricle. Both these cases were examples of the first form of fatty degeneration, or that in which the fatty matter seems primarily deposited on the surface of the heart.

CASE XXIX.—*Fatty condition of the Heart; Rupture of the left Ventricle; Free Oil in the Blood.*

"Margaret Newman, aged 90, died suddenly, having previously complained merely of debility and the infirmities that 'wait on age.'

"*Inspection twelve hours after death.*—The integuments covering the arms, thighs, and chest, presented large livid patches, and a crepitation was felt in the subcutaneous cellular tissue over almost all parts of the body, but particularly beneath the discoloured portions of the skin: the subcutaneous cellular tissue was likewise loaded with adipose substance of an unhealthy softness, pale and watery. Upon elevating the sternum, air was seen in the cellular tissue of the mediastinum; the pericardium was distended to the utmost, with blood partly fluid, partly in clots; the heart, thickly covered with adeps, particularly upon its posterior surface, was soft, pale, and flaccid, and globules of air were seen beneath its serous covering, arranged for the most part along the course of the coronary vessels.

"Near the centre of the anterior part of the left ventricle, there was a small lacerated opening, about a quarter of an inch in length: the substance of the ventricle was softened, most easily broken by the finger, and of a pale yellowish colour, as if infiltrated with purulent matter; the scalpel was greased in cutting the muscular substance, and upon the surface of the blood which had escaped from the divided vessels there floated numerous globules of oil.

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[1] Contributions to Pathological Anatomy. By R. W. Smith, A. M. Dublin Journal of Medical Science, First Series, vol. ix. p. 411.

"The abdominal viscera presented remarkable appearances: beneath the serous investment of the stomach, intestines, liver, spleen, and kidneys, air was extensively diffused; the liver was converted into a semifluid pulp, so much so that a stream of water poured upon it from a moderate height washed away the substance of the organ, the vascular structure alone being left; through this disorganized tissue air and oil were diffused; the spleen and kidneys presented similar appearances to the liver, and all these viscera, along with the stomach and heart, floated perfectly in water. Upon removing the liver from the body, the division of the vena cava gave exit to nearly a table-spoonful of a clear, perfectly transparent, limpid oil, followed by the blood of the vein. I collected about half an ounce of oil from what had escaped from the different organs; but nearly double the quantity might have been procured; several of the larger arteries were ossified; the brain presented no morbid appearance."

CASE XXX.—*Fatty condition of the Heart; Free Oil in the Blood.*

"A woman, aged 70, was admitted into the Richmond Hospital, having been found in the street, lying exposed to the wet and cold, and bearing the appearances of extreme poverty; she died about an hour after her admission.

"*Inspection eighteen hours after death.*—In the chest a considerable quantity of fluid occupied the cavity of the pleura, upon either side; the heart was remarkably soft, pale, and flaccid; its substance most easily broken, and its surface covered with a layer of fat, a quarter of an inch in depth; the parietes of the ventricles were thin; the surface of the blood was thickly covered with globules of limpid oil; the blood itself was thin, unhealthy in appearance, and without any disposition to coagulate; the vessels of the brain were greatly congested; the abdominal viscera healthy."

It will be proper, when we take a general view of the symptoms and history of this change in the heart's condition, to discuss the views of its general pathology which Dr. Smith has advanced in connexion with these cases. Let us, in the meantime, continue our examination of the cases observed in Dublin.

CASE XXXI.—*Long-continued Palpitation; Occasional and sudden Faintings; Sudden Death, with Apoplectic Symptoms; Extensive Fatty Deposits in the Heart.*

This case excited great interest in Dublin, and was communicated to the Pathological Society by Mr. Carmichael in the Session of 1840. The patient a clergyman, of upwards of sixty years of age, and of temperate habits, had generally enjoyed good health, and had suffered no inconvenience from any symptom referrible to the heart, to such a degree, as to confine him to his bed, or prevent him discharging his professional avocations. For many years, however, he had been subject to palpitations of the heart; and on one or two occasions he fainted without any assignable cause. The tendency to faint was on several occasions removed by the use of a small quantity of brandy or other stimulants. On the morning of his death he had performed the marriage ceremony between two members of his congregation, and was sitting at the wedding-breakfast when his head drooped, his breathing became stertorous, and in a few moments he was dead. The veins of the neck were turgid, and Dr. Hutton, who was present, opened the jugular vein, and tried every other means of resuscitation, but in vain. On the day following his death, the veins of the forehead became turgid, and yielded a sense of crepitation; and it was subsequently difficult to convince the friends of the deceased that he was actually dead, so much did the injection of the superficial vessels and the colour of the face simulate life. Dissection was performed five days after death. The cellular structure of the neck, upper extremities, trunk, and scrotum, was emphysematous. The scrotum had attained the size of a melon, and was almost transparent. Much fat existed under the abdominal integuments and in the anterior mediastinum.

The heart, covered with fat, was found with its right cavities greatly distended with air. The wall of the right ventricle was about two lines in thickness, and the muscle seemed nearly replaced by fat. What remained of muscle was greasy and friable. The left cavities presented similar appearances, though not to so great a degree. The liver, lungs, and brain were healthy.

In this case the turgescence of the veins observed at the time of death seems to favour the opinion of Dr. Adams, that death may in this disease be produced by venous congestion of the brain. This view was adopted by Mr. Carmichael, who considered that from the inability of the right ventricle to propel the blood through the lungs, the descending cava and veins of the head had become so overloaded that death was the result. He suggested that one small bleeding, followed by stimulants, should be the proper course in such attacks.

The remarkable turgescence of the subcutaneous veins giving, as in this case, the fallacious appearances of returning animation, has been well explained by Professor Smith. He refers it to the production of air, not only in the right side of the heart, but in the venous system generally, a change which takes place soon after death; and which produces the injection of the capillaries and veins of the surface.

The two following cases occurred under my own observation.

Case XXXII.—*Anæmic condition; Very slow Pulse, with valvular Murmur; Death, apparently from Syncope; Fatty Degeneration of the Heart, with Disease of the Aortic Orifice.*

A man, upwards of fifty years of age, was admitted, presenting much of the general characteristics of senile phthisis. His skin was of a pale yellowish tint, and his whole appearance indicated great debility. He complained of cough and dyspnœa, but did not refer any of his sufferings to the region of the heart. His pulse was generally 35 in the minute, though occasionally rising to 40. The action of the heart was regular, but feeble, and a valvular murmur with the first sound, precisely similar to that in mitral valve regurgitation, was always audible. This became louder on ascending the sternum, and was most intense on the right side, at the articulation of the second rib. We were inclined to consider this as an example of mitral valve disease, and supposed at first that the aortic murmur might result from anæmia. The patient died without any struggle. On dissection, the mitral valves were found healthy. The aortic valves were thickened and narrowed, but not permanently patent. Water poured into the aorta did not pass into the ventricle; the heart was soft and flabby, and, though not an example of complete fatty degeneration, was covered by a very thick adipose layer. The aorta presented several atheromatous patches.

In this case the second sound remained normal; there was no regurgitation into the ventricle. The valves were sufficiently diseased to cause a murmur with the first sound, but from their power of closing completely, the second was unaltered.

CASE XXXIII.—*Repeated pseudo-apoplectic attacks, not followed by Paralysis; Slow Pulse, with valvular Murmur, propagated into the Aorta.*

Edmund Butler, aged sixty-eight, was admitted into the Meath Hospital, February 9th, 1846. He stated that his health had been robust until about three years ago, at which time he was suddenly seized with a fainting fit, in which he would have fallen if he had not been supported. This occurred several times during the day, and always left him without any unpleasant effects. Since that time he has never been free from these attacks for any considerable length of time, and has had at least fifty such seizures. They are uncertain as to the period of their invasion, and very irregular as to their intensity, some being much milder and of shorter duration than others. They are induced by any circumstances tending to impede or oppress the heart's action, such as sudden exertion, distended stomach, or constipated bowels. There is little warning given of the approaching attack. He feels, he says, a lump first in the stomach, which passes up through the right side of the neck into the head, where it seems to explode and pass away with a loud noise resembling thunder, by which he is stupified. This is often accompanied by a fluttering sensation about the heart. He never was convulsed or had frothing at the mouth during the fit, but has occasionally injured his tongue.

The duration of the attack is seldom more than four or five minutes, and sometimes less; but during that time he is perfectly insensible. He suffered no unpleasant effects after the fits, nor had anything like paralysis. His last fit occurred about one month before admission. He has never heard it remarked that there was anything peculiar about his heart or pulse. At first he found that ardent spirits were the best restorative or prophylactic, but latterly he has not used them.

On admission, he was haggard and emaciated, but seemed the wreck of what was once a fine, robust man. He lay generally in a half drowsy state, but when spoken to was perfectly lively and intelligent.

He makes no complaint of his general health; his appetite is good, and he sleeps well; bowels regular, and the urinary functions are in good order. He has, however, some cough attended with a slight mucous expectoration. His intellectual powers are perfect. He complains of a feeling of chilliness over the body, and is never warm except when close to the fire. This has long been the case; and every day he gets a chill, generally in the afternoon, which is followed by an increased heat of the surface, but without sweating.

On percussion, the chest is universally resonant. The respiratory murmur loud, and combined, more especially posteriorly, with large mucous râles. The impulse of the heart is extremely slow, and of a dull, prolonged, heaving character, giving the idea of feeble as well as of slow action. The first sound is accompanied by a soft bellows murmur, which is prolonged into the commencement of the second sound, and is heard very distinctly along the sternum, and even in the carotid arteries. The second sound is also imperfect, though very slightly so; the imperfection being much more evident after some beats than others. Pulse 28 in the minute, of a prolonged, sluggish character; the arteries pulsate visibly all over the body, but no murmur is audible in them. They appear to be in a state of permanent distention: the temporal arteries ramifying under the scalp, just as they are seen in a well-injected subject. All the other cavities and viscera appear to be in a perfectly healthy state. Urine neither acid nor alkaline; of a light colour and clear; specific gravity, 1010; it does not afford a precipitate with nitric acid. He was ordered wine, and a liniment for the shoulder.

February 17th. The pulse has varied from 28 to 30 in the minute. The cardiac murmurs continue unchanged; that with the first sound is plainly audible over the upper part of the thorax, but is most evident along the course of the aorta.

His aspect and general health are greatly improved since his admission. He gets up every day, and is much stronger. The shoulder is almost quite well. The pulse has continued at about

28 or 30. He says he has had two threatenings of fits since his admission, both occurring in bed, *and both warded off by a peculiar manœuvre: as soon as he perceives symptoms of the approaching attack, he directly turns on his hands and knees, keeping his head low, and by this means, he says, he often averts what otherwise would end in an attack.*

We remarked to-day, on listening attentively to the heart's action, that there were occasional semi-beats between the regular contractions, very weak, unattended with impulse, and corresponding to a similar state of the pulse, which thus probably amounts to about 36 in the minute, the evident beats being only 28, so that there must be about eight of these semi-beats in the minute;—but these signs are very indistinct.

18th. He complains to-day of palpitation, and a feeling of uneasiness about the heart;—the impulse is increased, and is found to consist of two distinct pulsations. The murmur with the first sound is somewhat louder than before. On listening attentively, there are heard occasional abortive attempts at a contraction, probably about four in the minute. They do not destroy the regular intervals between the stronger sounds, but are heard, as it were, filling up the interval. We could not recognise a corresponding state of the pulse, which counted 32 in the minute.

In about three months this patient was again admitted into hospital. The cardiac phenomena remained as before, but a new symptom appeared, namely, a remarkable pulsation in the right jugular vein. This was most evident when the patient lay down. The number of the reflex pulsations was difficult to be established, but they were more than double the number of the manifest ventricular contractions. About every third pulsation was strong and sudden, and could be seen at a distance; the remaining waves were much less distinct, and some very minor ones could be perceived. These may have possibly corresponded with those imperfect contractions already noticed in the heart. The appearance of this patient's neck was very singular, and the pulsation of the veins such as we never before witnessed.

He has had scarcely any of the cardiac attacks since he was discharged; he referred the premonitory sensations to the right supra-clavicular region, but stated that he often experienced them without loss of consciousness having followed.

In a clinical point of view, we may separate cases of fatty disease of the heart into two classes. In the one the alteration is found in various degrees of development, although other organs than the heart have been prominently affected; in the other, the heart affection seems the principal lesion, and the general health continues good. In some of the first class the symptoms may be so modified by the cardiac disease as to lead to the latter being suspected during life, yet in others the condition of the heart is only recognised on dissection, or, it may be, microscopic examination. The memoirs of Dr. Ormerod and of Dr. Quain contain numerous examples of this kind, and show that in many chronic diseases this condition of the heart is in progress, although its existence is commonly overlooked or unsuspected.

In this way the disease is met associated with phthisis—with bronchial disease—chronic affections of the liver and kidneys—diseased prostate gland, chronic rheumatism, and under various conditions of the gouty state. In other cases, too, this lesion may exist, though not to such a degree as to draw special attention. And it is, doubtless, the cause why so many patients labouring under various chronic affections sink rapidly when placed on a restricted regimen, or when kept too much under the influence of lowering medicines. The recognition of this class of cases is, perhaps, of more importance than that in which the disease is prominent, and, as it were, isolated.

We owe not only the statement but the best illustrations of this important clinical fact to Dr. Ormerod, whose memoir is of great value,[1] not only as to the history of the disease, but its

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[1] Observations on the Clinical History and Pathology of one form of Fatty Degeneration of the Heart. London Medical Gazette, vol. ix. p. 739.

The researches of Drs. Paget, Ormerod and Quain, of Hasse, and other Pathological anatomists, have given us full information as to the microscopical appearances in this disease. But as the account given by Dr. Ormerod is the most succinct and best adapted to give a proper idea of the affection, I shall quote it here.

"To the unassisted eye," says Dr. Ormerod, "the muscular substance of a healthy heart presents characters distinguishing it from ordinary muscular tissue; for it is more compact and homogeneous, and not loosely divided into bundles of fibres, as is ordinary muscle. Under the microscope it also presents some striking differences, the transverse striæ being less distinctly marked, and the fibres having a singularly granular appearance. It is very important to notice this normal difference at the outset; for the first step towards fatty degeneration consists in the loss of the continuity of the transverse striæ, and in the increase microscopic anatomy. And he has shown that without the employment of the microscope it will not be safe to assert that the

of this granular marking of the fibres, which would seem to be in some degree their normal appearance.

"This is the first step, and, as wholly undiscoverable by the naked eye, may often pass unnoticed, unless something in the symptoms, or some change in the general condition of the heart, call particular attention to that organ. Such conditions may be a small, pale, flabby state of the heart, not inaptly compared to the colour of withered leaves, and to the feel of a moist glove. But such are not commonly the signs which call attention to the existence of this structural change; they are ordinarily much more obvious.

"On opening a heart thus affected, the interior of the ventricles appears to be mottled over with buff-coloured spots of a singular zigzag form. The same may be noticed beneath the pericardium also; and, in extreme cases, the same appearance is found, on dissection, to pervade the whole thickness of the walls of the ventricle and of the carneæ columnæ. Of these latter, the musculi papillares seem most liable to be affected; not to say that this form of disease never occurs in the walls of the auricles,—at least I have never seen it there.

"Microscopic examination reveals the nature of these spots; they are not deposits, but distinctly degenerated muscular fibres. The outline, not merely of the masses, but of each single fibril, is accurately preserved. Instead, however, of transverse striæ and nuclei, the evidences of active vitality, there is little to be seen but a congeries of oil globules. The whole history of the degeneration may be traced in one of these little spots. First, from the immediate neighbourhood of the spot we may obtain healthy muscular fibre; then the transverse striæ become less distinct; they are rows of dots rather than continuous lines; then the intervals between the dots become wider, and the dots themselves run into longitudinal rather than transverse lines; and then all the regularity is lost, and the dots appear to stud the surface all over, like the points on a bit of fish-skin. Probably long before this time the fibre has lost all its properties as a muscle; but there are further changes to observe; for now, mixed with these minute dots, are to be seen small oil globules, which increase and coalesce till the fibril presents little else but a congeries of oil drops contained within the sarcolemma.

"This is not the only change which the fibres undergo; for, with whatever care they are disintegrated, they are found to be short, and as if unusually brittle,—a general condition which may, perhaps, be of more serious importance than the actual fatty degeneration of the organ.

"Such are the most common features of the disease, and sufficiently obvious when really once noticed, to prevent their being readily overlooked afterwards. But we must not rely too exclusively upon them; for, as already observed, in the absence of these little spots marking the extreme degree of fatty degeneration in single points, the disease may have pervaded the whole substance of the heart; and the recognition of such a change will be difficult in exact proportion to its extent, and, therefore, its importance, from the want of healthy tissue wherewith to contrast the diseased fibres. And there is no solution for the difficulty except in the use of the microscope, whose information, should anything casually induce us to solicit it on this subject, at least is infallible."

heart is free from this disease. Besides giving examples of the affection in its well-developed and manifest form, he has recorded cases of various degrees of the fatty change in the following diseases:—delirium tremens; paraplegia; dropsy; hydrothorax; bronchitis; marasmus; epistaxis; hemorrhage from placenta prævia; acute and chronic phthisis; valvular disease; encephaloid disease of the pericardium; renal disease; pneumonia; apoplexy; and fever. Of these facts the practitioner should take especial note, not that he is to believe that the fatty state of the heart was the cause of these various maladies, or produced by them, but that it is a frequent, most important, and often latent complication in chronic disease; and it behooves him to make this knowledge available in his treatment and prognosis.

He must bear in mind that in many chronic cases, even although there be no symptom or well-marked sign to draw attention to the heart, yet that it may be more or less affected with this disease; and that although the circulation appears to be carried on with a fair amount of strength, yet that the muscular fibres of the heart may be atrophied, and under these circumstances liable to a sudden failure of action.

In the treatment, then, of many chronic affections, and, above all, in that of acute irritations supervening upon chronic disease of any kind, or occurring in persons past the prime of life, or again, in younger patients whose systems have been, from whatever cause, debilitated, it becomes necessary to take the state of the heart into consideration, and by every means in our power to determine how far its vital and organic conditions have been affected: for there is no class of patients in which a depraved hæmatosis has occurred, from deficient innervation or nutrition, on the one hand, or from excess of nutrition, on the other, that is not liable to the disease.

We may inquire whether this disease is to be considered as a primary local lesion, or one secondary to certain changes in the blood itself. Without going into nice distinctions, but looking at the matter practically, we may believe that both forms of fatty disease, namely, the growth of fat upon the organ, and the original degeneration of the muscular fibre itself, are to be referred to general conditions of the system. The first form is the result of circumstances which favour the formation of fat, while its amount is still within the limits of health; and the second, or the fatty transformation, is commonly met associated with fatty disease in other parts, and may be safely held as secondary to a general condition. Dr. Quain believes "that the molecular fatty matter in the fibre is the result of a chemical or physical change in the composition of the muscle itself, independent of those processes which we call vital."[1]

Reasoning from facts observed with reference to the formation of adipocire in dead animal matter, this author comes to the conclusion that when the protein compounds, albumen and fibrine, are placed in a position unfavourable to their organization, or when they enter into the composition of tissues whose organization or vitality is imperfect, they themselves degenerate and pass into fatty matter. The change, according to him, is chemical, and is induced by whatever tends to weaken those vital powers which preside over the nutrition of the organ. This opinion seems to have been hinted at by Rokitansky.

But what is the immediate cause of this deficient innervation of the heart? This question is of importance if we consider the affection as a local one. Dr. Quain holds that obstruction of the coronary arteries is common in this affection; and, without affirming or denying this proposition, we may believe that this lesion might produce the disease. Yet examples are to be met with of fatty heart in which no such condition exists. And the question arises—Was the disease of the arteries but one of that series of changes which induced the general disease of the heart? Again, ossification of both the coronary arteries may exist, and yet the muscular structure be found not only without atrophy, but red, firm, and in all respects healthy; nay, further, the left ventricle may be hypertrophied, as in cases where the orifice of the aorta is permanently patent, while the coronary arteries are obstructed. The occurrence of fatty heart, as a sequel to pericarditis and endocarditis, is noticed both by Dr. Williams and by Rokitansky; and it is probable that this form may be the best example of the disease occurring as a local affection. In the case of pericarditis, two circumstances would favour its development. There is probably a case in which the plastic matter takes on the fatty transformation, so as to cover the heart with a layer of fat, which subsequently increases under the law of elective affinity. Again, it may be that from the effect of the adhesion, atrophy and degeneration of the muscular fibre are produced; thus both forms of fatty heart may occur in the same individual, and the case be originally an example of local disease.

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[1] On Fatty Disease of the Heart. By Richard Quain, M. D. Medico-Chirurgical Transactions, vol. xxxiii. p. 140.

I have already spoken of the effect of adherent pericardium in producing atrophy of the heart. But in disease, many paths conduct to the same end; and the duty of the physician is, first, to learn the mode of recognising the affection, no matter how produced, and next, to ascertain its various causes. So far back as 1836, Professor Smith showed that, in certain cases of fatty heart, free and limpid oil existed in large quantities in the blood, a condition in which, as might be expected, other organs besides the heart were found degenerated. This appears to prove that a fatty state of the heart may be caused not alone by degeneration of the protein compounds, but also from oil already formed and circulating in the blood itself.

GENERAL DIAGNOSIS OF THE DISEASE.

If it be inquired how far we have gone, since the time of Laennec, in establishing the diagnosis of this affection, it will appear that as yet but little has been done. Laennec declared that he knew of no means by which the diagnosis of fatty degeneration of the heart could be made; and Dr. Ormerod, writing in 1849, observes, that "the most extreme cases detailed may show that the diagnosis on general or physical grounds is almost impossible." "We cannot," he says in another place, "predict with certainty in any case that we shall find this lesion after death; but it is difficult for any pathological observer not to be led to suspect the existence of a disease in the repetition of the same circumstances under which he has seen it occur previously."

The diagnosis of this condition is not only possible but often free from difficulty, at least where the disease is confirmed. On the other hand, minor degrees of the affection are to be determined less by direct signs than by some general characters.

The diagnosis turns upon three points:—

1. The existence of physical signs and symptoms of diminished force of the heart.

2. The occurrence of certain symptoms, principally referrible to the brain, which indicate either anæmia on the arterial, or congestion on the venous side, of the cerebral circulation.

3. Symptoms referrible to the respiratory function, which appear to arise from deficient power in the right ventricle.

It is still to be determined how far we can distinguish during life the cases of weakened and dilated hearts, such as have been already described, from those of fatty degeneration. Microscopical anatomy shows that in many of the former class there is more or less of the adipose deposit. And it is plain that to the practical physician there is a relation between the diseases; for similar exciting causes concur in their production, and in both the effect of the disease is traceable to the same vital condition, namely, debility of the heart.

In its higher degrees of development this affection is most frequently met with in persons who have passed the prime of life; but minor shades of it occur in young patients, especially where there is a complication with other visceral diseases, as, for example, pulmonary tubercle. On the other hand, some of the most remarkable instances are found in very old and long bedridden subjects; and it is observed that in such cases the alteration is not confined to the heart, but extends also to the voluntary muscles, and even to the skeleton, producing atrophy and fragility of the bones, with a great deposit of oily matter in the cavities and cancelli of the osseous tissue.[1] Though varying and apparently opposite, its exciting causes are generally reducible to those which would induce a depraved hæmatosis. The over-fed and luxurious, on the one hand, and the victim of want, on the other, are liable to the disease.

Although complication with various local diseases, or with a special morbid state, such as gout, is not uncommon, yet judging from the good state of the general health, and the absence of lesion in the digestive, respiratory, and nervous systems after death, we must admit that the fatty heart may be, in a large number of cases, practically considered as a local affection.

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[1] Of this condition numerous specimens may be seen in the Museum of the Richmond Hospital.

It is probable that in these uncomplicated examples, the disease attains its greatest development, and exhibits the most characteristic symptoms.

The symptoms may be divided into those referrible to the nervous, respiratory, and circulating systems.

Of the nervous symptoms, the most important are the attacks of apoplexy, or pseudo-apoplexy, to which these patients are so liable. This affection differs from ordinary sanguineous apoplexy in three particulars, namely, the frequent repetition of the seizures, the rarity of consequent paralysis, and the fact that there is not only danger from an antiphlogistic treatment, but benefit, both remedial and preventive, from the use of stimulants.

In some cases the character of these attacks approaches to that of syncope; and it is difficult to say how much of the affection is produced by the want of arterial, or the stasis of venous blood. In the earlier periods of the case the attack is more of syncope, in the later it becomes apoplectic. The attacks may occur without warning, and the first seizure be fatal. This, however, is rare. In most cases there are numerous seizures at irregular intervals; and in some, sensations referrible to the epigastrium and head, having a resemblance to the epileptic aura, give notice to the patient that he is about to be attacked. In some there is a momentary unsteadiness in walking, and in others a tendency to faint, which may be dissipated by any ordinary stimulus; while in the more decided cases the patient becomes suddenly comatose, a condition which may be preceded by loss of memory and a lethargic state. I have at present under my care a patient whose earlier attacks were syncopal; they are now apoplectic, and come on during sleep, each one being preceded by a slight convulsion. On recovery, and after all the comatose symptoms have passed away, he remains for half an hour or an hour unable to recognise his most intimate friends and relations; even his wife he has mistaken for his mother. This patient is 63 years of age. This latter symptom has been observed in a case of weak heart which lately occurred in Dublin; the patient frequently failing to recognise friends who had been his intimates for half a century. The duration of the attack is generally short, paralysis is rare, and when it occurs does not seem referrible to any anatomical lesion of the brain.

The question as to whether these singular attacks are dependent upon deficient arterial supply or rather upon venous congestion, is a difficult one, but it does not involve any important point of practice. It is true, that whatever arrests the action of the heart will retard the flow of blood in the veins of the head, but it could not cause a state of hyperæmia. The opinion that the apoplectic seizures are owing to deficient arterial supply seems the most tenable. The suddenness of the attack, and, in many instances, the rapidity of the recovery, are in favour of this view. I have noticed one case in which, on the occurrence of the premonitory symptoms, the patient, by hanging his head so that it rested on the floor, used to save himself from an attack. A case lately occurred to me of an aneurism of the aorta, in which three successive ruptures of the sac took place, with intervals of several days. Each rush of blood was attended with the best-marked syncopal coma and convulsions. Finally, dissection does not show any extraordinary congestion of the brain; and we learn from auscultation that the action of the heart is feeble.

This view of the cause of the attacks appears to be still further corroborated by the occurrence of symptoms of a similar nature in the case of dilated mitral opening by Dr. Fleming, which has been already given. Here the ventricle was hypertrophied to a great degree, but the patient suffered from regurgitation into the left auricle.[1]

We can, therefore, only adopt in part the plan of treatment suggested by the late Mr. Carmichael, which was to relieve the vessels of the head by venesection, while at the same time stimulants should be used to excite the action of the left ventricle.

Symptoms referrible to the respiratory function.—There is no evidence that the existence of this disease, even in an aggravated form, is an exciting cause of any organic affection of the lung. On the other hand, the researches of Ormerod, Quain, and others, have demonstrated the frequent combination of fatty heart with pulmonary disease; but in such cases we may hold that the conditions of the lung and heart have little, if any, mutual relation; they are rather to be considered as the secondary accidents of a general morbid state.

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[1] See Case XXIII. page 222.

But there is a symptom which appears to belong to a weakened state of the heart, and which, therefore, may be looked for in many cases of the fatty degeneration. I have never seen it except in examples of that disease. The symptom in question was observed by Dr. Cheyne, although he did not connect it with the special lesion of the heart.[1] It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations. This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient. I do not know any more remarkable or characteristic phenomena than those presented in this condition, whether we view the long-continued cessation of breathing, yet without any suffering on the part of the patient, or the maximum point of the series of inspirations, when the head is thrown back, the shoulders raised, and every muscle of inspiration thrown into the most violent action; yet all this without râle or any sign of mechanical obstruction. The vesicular murmur becomes gradually louder, and at the height of the paroxysm is intensely puerile.

The decline in the length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnœa occurs. This is at last broken by the faintest possible inspiration; the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale.

In other cases we see the symptom of sighing to occur in a different manner: at irregular intervals the patient draws a single deep sigh, especially when he suffers from fatigue, want of food, or of his ordinary stimulants. This is the commonest form of the affection.[1] In one case it was always most evident when the patient was lying down.

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[1] See page 319.

The phenomena of circulation are next to be considered.

We are in want of a sufficient number of observations to enable us to declare whether in the earlier periods there is any marked character of pulse as to strength, frequency, or regularity. Many of the recorded cases of the minor stages of the disease are deficient in accurate observations of the pulse; but it may be held that no special character of pulse has been established. In some the pulse has been weak, rapid, and irregular; in others it does not seem to have differed materially from that of health.[1] But in confirmed cases we may meet with three important characters of pulse:—

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[1] The sighing respiration may be observed in persons who are labouring under certain forms of gastric or hepatic derangement, and is occasionally a symptom of undeveloped gout. It disappears under appropriate treatment, and probably indicates a temporary weakness of the heart. I lately saw a case of long-continued sighing, in which it had apparently arisen from depression and anxiety of mind, but had, as it were, become a habit. The patient was a lady of very nervous disposition. A feeble murmur attended the first sound of the heart. In this case there was probably no organic lesion, for the symptom had long existed, and there were no signs of progressive disease.

Sufficient attention has not as yet been directed to this character of respiration. It is, when confirmed, almost pathognomonic of a weak and, in all probability, a fatty heart; but whether it is to be taken as indicative of the predominance of the fatty change on the right side of the heart is still an open question. Laennec has described a form of asthma with puerile respiration, and he attributes the disease and the signs to some special modification of the nervous influence. He observes, that he has never met with it except in persons affected with mucous catarrh, and holds that dyspnœa, arising from the mere increase of the natural want of the system for respiration, can never amount to asthma without the catarrhal complication. But he further speaks of adults and old persons who have puerile respiration without catarrh, and who, though they are not, properly speaking, asthmatic, are short-breathed and liable to dyspnœa on the slightest exercise.

It is possible that in some of these cases at least, the heart may be in an incipient stage of fatty degeneration. I have observed the symptom in a gentleman of about 70 years of age, who has many symptoms of a weak heart. The action of that organ is regular, but the impulse is extremely feeble, and the pulse compressible. The sounds, especially the first, are very indistinct; there is no bronchial râle, but well marked puerility of respiration exists over every portion of the thorax. He principally complains of dyspnœa on exercise, or on any mental agitation; and the symptoms have only become prominent within the last eighteen months. So far as the permanent condition of the respiration is concerned, this case answers perfectly to Laennec's description of dyspnœa with puerile respiration. See Dr. Forbes's translation of the work of Laennec,—Article, Asthma with Puerile Respiration.

1. The pulse somewhat accelerated, but occasionally intermitting; its strength may be but little altered.

2. The extremely weak, rapid, irregular, and tingling pulse, (*pulsus formicans.*)

3. The permanently slow pulse, the rate of which varies from 50 to 30 in the minute, or even less.

It is probable, that in the third class of cases, or those with a permanently slow, though distinct and regular pulse, the disease has either advanced to a great degree, or has at all events affected the different portions of the heart equably; and that we may attribute the weak and irregular pulse to conditions of the heart in which only certain portions of the organ have degenerated, or where there is a great difference between the right and left sides of the organ. It is further probable that the heart may be in two very different conditions previous to the commencement of the fatty change; and that in the case with irregular pulse, a merely weakened and perhaps dilated condition has preceded the deposit of fat globules in the muscular fibre; while in the third class the change has occurred without previous alteration in the structure or mode of action of the heart. Some of the cases observed in persons who have been long bed-ridden, and who have died from rupture of the left ventricle, are of this description. Additional observations, however, are necessary to elucidate this subject.

If we inquire whether irregularity of pulse is indicative of valvular disease in this affection, we must consider that the symptom may be met with in cases of weak, dilated hearts, without valvular disease, and, therefore, that we might expect it in the fatty degeneration. On the other hand, the occurrence of cases with a perfectly regular though slow pulse is a remarkable fact. In well-marked cases, where irregularity, rapidity, and smallness of pulse exist, we ought not, even though there be no valvular murmur, to declare too strongly against the existence of valvular obstruction; bearing in mind, first, that the very weakness of the heart may prevent the appearance of murmur; and next, that valvular disease is a not infrequent combination with fatty heart. In most of the cases which I have seen, this valvular affection was at the aortic orifice, and the pulse was slow and regular. The following case exemplifies the disease with a contracted mitral orifice.

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[1] This circumstance is worthy of consideration in connexion with that which I have recorded as occurring in cases of the softening of the heart in typhus, in many of which the pulse is quite a fallacious guide in determining the strength of the left ventricle.

CASE XXXIV.—*Fatty degeneration of the Heart; Contraction of the mitral opening; Valvular Murmur loudest at the Apex; Feebleness, irregularity, and rapidity of Pulse.*

A man, aged 50, who had gone through a long period of suffering and want, was admitted into the Meath Hospital in a state of great debility. His pulse was small, rapid, feeble, and irregular, no two successive beats having the same character; sometimes it was short and momentary, then more distinct and, as it were, longer; while at others it had the creeping, tingling character (*pulsus formicans.*) He was liable to sudden feelings of approaching death, attended with temporary loss of recollection, and was affected with frequent and deep sighing, especially when in the recumbent posture. The lungs showed signs of chronic bronchitis, and the liver appeared engorged. On each attack of the pulmonary dyspnœa the liver became augmented in volume. The impulse was very feeble, and a valvular murmur existed loudest at the apex and over the left side of the heart.

On dissection, the heart was found generally enlarged, and covered with a thick layer of fat, lying between the muscular structure and pericardium, and in many places dipping down through the fibres. It was most abundant at the base and the upper and anterior portions of the right ventricle. The mitral opening presented all the characters of the crescentic slit as described by Dr. Adams.

We may divide cases of this affection into those uncomplicated with disease of the valves or aorta, and those in which either or both of these combinations exist. The fatty degeneration, considered alone, does not, so far as we know, afford any special or separate sign; and it would be difficult or impossible to draw a line of distinction between the signs of simply weakened heart and of this condition combined with fatty deposit. The feeble impulse, the altered rhythm, the irregular action, the shortened first sound, and the often unanalyzable character of the heart's sounds, appear common to both; and it is certain that in many minor cases of this condition, particularly when it is combined with other diseases, such as pulmonary tubercle or lesion of the liver and kidney, the heart's sounds may present no abnormal character.

In extreme cases we should expect that the impulse and first sound of the heart would be wanting or greatly diminished, just as is observed in the typhoid softening; but there is a great want of recorded observation on this point. In a case recently under my care, of an extremely weak and semi-fatty heart, where the patient was sufficiently thin to allow us accurately to observe the impulse of the organ, I found that it displayed a novel character. The impulsive force seemed equably diffused over the whole ventricular region; there was no striking of the apex against the ribs, so that it could be distinguished from the remainder of the impulse; the ventricle, in fact, seemed passive, and the character of the entire impulse closely resembled that of an aneurism with thin parietes, but with a feeble pulsation. This I have already noticed.

But where the disease is combined with lesion of the aortic valves, the diagnosis is not difficult, for then we have a combination of symptoms and signs which, as far as we know, is not met with in any other case. The first sound of the heart, as a muscular sound, is scarcely distinguishable, especially over the left ventricle, but it is replaced by a prolonged murmur, loudest at the base of the heart, and extending at least to the commencement of the descending aorta. Notwithstanding the existence of this sign, the second sound remains unaffected, so that we have here, so far as the heart is concerned, the signs of mitral disease, and on the side of the aorta, those of anæmia or chlorosis; yet the disease is neither one nor the other, but consists in the combination of a fatty left ventricle with alteration of the aortic valves, of such a nature that though a murmur is produced during the exit of the blood, there is no regurgitant sign, the valves being competent to close the orifice. In such cases, the ascending aorta is often found studded with patches of atheroma.

In a memoir on "Slow Pulse" I have shown of what importance the combination of disease of the aortic orifice with a fatty state of the left ventricle is in the diagnosis of this disease, inasmuch as we have then produced that particular group of signs and symptoms which is almost pathognomonic. In this condition the first or systolic sound is masked by, or it may be lost in, the valvular murmur which is propagated into the aorta and carotids, and which is single. There is no regurgitant murmur, and the second sound remains distinct. Now, if we confine ourselves to physical signs alone, it may be laid down that murmur with the first sound, distinct at the left side,—a single murmur propagated into the great vessels,—or, it may be, also developed in these vessels, and co-existing with a perfect second sound, is met with in but two cases, namely, the chlorotic condition and the disease under consideration. But the differential diagnosis is easy, for the sex, age, history, and vital symptoms, will give abundant data to enable us to say whether the case is a fatty heart with aortic disease, or whether the phenomena arise from an imperfect state of the blood itself. It is possible, that in the fatty disease in advanced life, with organic change of the orifice, anæmia may have some effect in causing these murmurs. It may assist in their production; but in the young female it is most unlikely that ossification or atheroma of the aorta assists the anæmic condition in producing the signs in question.

There is, however, reason to believe that a case may be met with in which the signs are so modified as that even the second sound disappears, and yet the evidences of regurgitation are wanting. This condition is one of the most remarkable that can be met with, and its explanation is difficult indeed. I have often observed a diminution of the second sound, but its complete extinction in a patient who had the symptoms well marked of a fatty state of the heart must be at present considered as a rare occurrence. The following case is worthy of study.

A gentleman, aged 63, of a strong and healthy habit, suffered from an attack of rheumatic fever about eleven years before I saw him. He recovered, and continued in good health for seven years.

I could not ascertain whether the heart had been engaged during the fever. About four years since, he became liable to fainting fits, at long intervals of time, between which his health continued good. In some of the attacks the fainting was so complete that he would fall from his chair; but at other times they were shown by a tendency to faint rather than by actual syncope. On the occurrence of flatulent eructation and sickness of stomach, the attack would pass away.

During the last six weeks, however, the symptoms became more formidable. The attacks came on generally at night and while the patient was asleep; and his wife was made aware of their approach by observing that his respirations became gradually so feeble and low that they could be hardly perceived. In this state, if he was not roused, a slight convulsion occurred; and it sometimes happened that it was by the sudden motions of the arms and legs that his wife was awakened. The hands and legs were then found cold, and the respiration was of the puffing kind often observed in coma. There was no stertor; and it is a remarkable fact that this gentleman, up to within the last six weeks, was accustomed always to snore during sleep, but since that period he sleeps without snoring. Epistaxis to a slight degree has occurred more than once in these attacks, which last about three-quarters of an hour. His recovery is slow, and attended with confusion and dulness of intellect. It has been always remarked, too, that during the attack his breath, which is naturally sweet, has a heavy and offensive odour.

The impulse of the heart is feeble, but somewhat extended; pulse about 80, with tolerable strength and natural fulness, but without any throbbing character. It intermits at about every tenth or twelfth beat. There is no visible throbbing of the radial or carotid arteries.

On applying the ear nothing is heard but a single slightly musical bellows murmur attending the systole of the heart, and becoming even more distinct in the course of the aorta and the carotids. It may be found also in the interscapular region, where it is feebly heard at the end of expiration. No trace either of the first or second sound of the heart can be found except at the moment immediately following the intermission, when one short, indistinct, systolic sound may be perceived. The want of the sounds of the heart is as complete over the right as the left cavities.

That this case is one of fatty degeneration of the heart cannot be doubted; and the diagnosis rests upon the same general grounds as in the former cases, namely, the existence of symptoms and signs of a weak heart in connexion with evidence of disease of the aortic orifice. But I have never before observed the extinction of the second sound, unless attended with the signs of regurgitation; and future observations alone can be expected to explain why, with a single systolic murmur, the second sound should cease.

The list of symptoms and signs will then stand as follows:—

1. Repeated attacks of syncope, and of pseudo-apoplexy.
2. Permanently slow, or irregular and feeble pulse.
3. A feeble impulse, with bellows murmur accompanying or replacing the first sound.
4. A murmur existing in the aorta, not of the regurgitant character. The second sound healthy.
5. Both sounds absent, and replaced by a bellows murmur, not regurgitant.

But it must be remembered that this group belongs to the fully formed disease. In this, as in most other chronic affections, the physician is often called on to give an opinion in cases where the disease is only commencing, or has not yet reached its full development; such cases are by no means rare. Under these circumstances the following conditions may occur:—

1. The general health being good, the patient complains of dyspnœa on exertion, and will often refer the source of difficulty to the epigastric or cardiac region.
2. He may have nocturnal attacks of orthopnœa, coming on after some hours of repose in the horizontal position. They begin with a slight feeling of oppression, which gradually increases to orthopnœa.
3. The pulse may not be either irregular or unusually slow; it may have no particular morbid character; or may show, as it were, a shade of the character of the regurgitant pulse of permanently patent aortic orifice.
4. He may have occasional suspensions of the respiratory act, yet without suffering, and his sensations are as if he were able to live without breathing.

5. A certain degree of indolent enlargement of the liver may exist.

6. The heart's action is somewhat peculiar, giving a heavy, sluggish throb, which apparently impinges on a large surface. I have seen a few cases in which the sound of the left ventricle was certainly diminished, while the second sound remained unaffected.

7. In such cases we may have a slight and, as it were, aborted murmur at the situation of the aortic valves, but which is not propagated into the arteries. It is a single sound.

I have recently seen a case exemplifying these observations. It was that of a gentleman of advanced age, who had for some years changed his mode of living, and given up his habits of active exercise in the open air. He had been always strictly temperate. The liver was enlarged to a slight degree, and he at one time was extremely apprehensive of any sudden pressure or blow on the abdomen, as he had the feeling that any forcible pressure on the belly would cause his death. His digestive and nervous functions seemed quite unimpaired, but though he had not lost flesh, his appearance was certainly anæmic.

In considering the various forms of weakened heart, it becomes obvious that, although the organic condition of the heart may be different, considered in a purely anatomical point of view, yet its dynamical state is much the same, and that the injurious effects are traceable to the diminished force or energy of one or both sides of the heart. The attacks of syncope, those of the pseudo-apoplexy, and the occurrence of sudden death, are traceable to the weakness of the left ventricle, no matter whether that weakness be simply muscular debility without fatty deposition, or whether it occurs in connexion with that change. The slowness of the pulse seems to imply an equally weakened state of both ventricles; while its irregularity and inequality may be attributed to imperfect contractions of the heart, or to a want of consent between the ventricles.

Again, the fulness of the liver, the tendency to anasarca, and, above all, the attacks of cardiac asthma, and of the peculiar ascending and descending respirations, alternating with periods of apnœa,—all point out a weakened condition of the right cavities; and it finally appears probable, that the good effects of tonic and stimulating treatment, in that form of hypertrophy connected with permanent patency of the aortic valves which has been dwelt on by Dr. Corrigan, is to be explained by assuming that with the hyertrophy there is a weakened condition, with or without the deposition of fat. We have seen that disease of the aortic valves is a not uncommon complication of the fatty heart; and though in our cases the valve was still competent to close the orifice, it is probable, had the patients lived long enough, that the destruction of the valvular character of the orifice would have been complete.

Before proceeding to recapitulate the facts which have been now stated, I am anxious to correct an error into which Dr. Quain has fallen with respect to my views as to the cause of the slowness of the pulse in this disease. In his elaborate Memoir on Fatty Diseases of the Heart, Dr. Quain has the following observations:—

"The slowness of the pulse is sometimes quite remarkable. In one case (No. VI. Series I.) the pulse was as slow as 24 in a minute when the patient was lying down, and never above 32. There is an interesting communication by Dr. Stokes in the Dublin Journal (August, 1846, p. 73,) in which he seeks to show the connexion between slow pulse and disease of the aorta or its orifice. I am disposed to think that the soft, flabby, feeble, if not fatty state of the heart, which was present in all his cases, is more intimately connected with the state of the pulse than the aortic disease, which does not appear to have assumed a constant or uniform character in any of the cases."

It is hardly necessary for me to observe, that I never thought of attributing slowness of the pulse to disease of the aorta or its valves. The object of my paper was to show that the frequent combination of disease of the aortic orifice with fatty degeneration of the heart might be made to assist in the diagnosis of the latter affection, inasmuch as under these circumstances there was produced a group of symptoms and signs having a special character, namely, the combination of slow pulse, pseudo-apoplectic attacks, and murmur propagated into the aorta, while the second sound remained clear. In fact, my observations were based upon, and intended to illustrate, the views of Dr. Adams on fatty degeneration of the heart.

RECAPITULATION.

1. That the two forms of fatty disease of the heart, as indicated by Laennec, may be admitted.

2. That although we cannot as yet distinguish these forms during life, yet that in confirmed cases the disease is not difficult of detection.

3. That the first of these forms, or that in which fat is deposited on the surface of the heart, is the most frequent, and may occur in patients who are the subjects of other chronic diseases, and who are not advanced in life.

4. That the second form, or that in which there appears to be a primary degeneration of the muscular fibre, is commonly found best marked in the very old and bed-ridden subjects.

5. That it is in this form that rupture of the heart is most liable to occur.

6. That between the cases of this disease in its first form, and those of simple weakness with dilatation of the heart, it is often difficult to draw the line of distinction.

7. That it may occur as a complicated affection, or associated with various chronic diseases, of which the most common appear to be chronic tubercular affections of the lung; hepatic or renal disease; and, lastly, atheromatous alterations of the aorta.

8. That though often associated with the gouty state, it may occur independently of that condition.

9. That the disease may, on the one hand, affect a heart already in a state of hypertrophy, or, on the other, of atrophy.

10. That in its earlier stages but little change is apparent to the unassisted eye in the anatomical condition of the ventricle; and it is by microscopical examination alone that we can determine the actual freedom of the organ from disease.

11. That with reference to the condition of the valves, we may divide the cases into three classes, namely, those in which there is disease of the aortic orifice; those where the mitral opening has been narrowed; and, lastly, those in which there is no organic change in the valves.

12. That in the first series of these cases there is probably a

more close connexion between the valvular disease and the fatty condition of the heart than in the second.

13. That obstruction of the coronary arteries is often met with in this disease, and that in certain cases it may assist in the production of atrophy and the fatty state of the heart; but that we may often look on this condition as one of the adjuncts rather than the primary cause of the disease.

14. That fatty degeneration of the heart may be met with, and yet the coronary arteries be found unaffected.

15. That although it may occur in hearts which have been damaged by inflammation, yet that it is seen in cases where the organ has never been thus attacked.

16. That in certain cases of the disease, oil in a free state is found in the blood.

17. That in such instances many other organs are found altered and degenerated, especially the liver and the bony structures.

18. That the pulse presents various characters: it may be natural, in strength and frequency; it may be large, soft, regular, and slightly collapsing. It may be irregular and unequal, and intermitting; it may be permanently slow, or altogether disappear for a length of time before death.

19. Cerebral symptoms of a remarkable character, are commonly present in this disease. These consist in the occurrence of repeated pseudo-apoplectic attacks, of various degrees of intensity and duration. They are seldom followed by paralysis. Attacks of vertigo, dimness of vision, and syncope, are observed.

20. That the frequency and severity of these attacks seem to be increased by whatever exhausts or lowers the patient, and to be diminished by the use of tonics and stimulants.

21. That sudden death, without rupture of the heart, or solution of continuity of the brain, is liable to occur in this disease.

22. That a remarkable sensation of sinking of the heart is frequently experienced.

23. That the formation, after death, of air within the cavities of the heart and in the veins may produce the fallacious appearance of returning life.

24. That in such cases the cadaveric pneumatosis of the heart may cause a singular inflation of the organ.

25. That the respiratory symptoms are divisible into three classes:—

a. Attacks of dyspnœa on exertion, similar to those observed in other cardiac diseases.

b. Gradually increasing difficulty of breathing, amounting to orthopnœa, and coming on spontaneously.

c. A form of respiratory distress, peculiar to this affection, consisting of a period of apparently perfect apnœa, succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the commencement of another apnœal period. During the height of the paroxysm the vesicular murmur becomes intensely puerile.

26. That although this affection may exist without valvular disease, yet the co-existence of a certain amount of alteration of the aortic valves is common; so that the combination of a slow pulse, a feeble impulse, and a diminished first sound over the left ventricle, attended with a single murmur, while the second sound remains clear, will be sufficient for the diagnosis of the disease in many cases.

27. But that in cases where no such murmur exists, we may also diagnose the disease when we observe,—in connexion with a slow and regular, or rapid, but irregular and unequal, pulse,—the occurrence of the pseudo-apoplectic symptoms, with or without the special character of apnœal intervals and the ascending and descending respirations.

APPENDIX TO THE PRECEDING CHAPTER.

General Diagnosis.—The diagnosis of this affection has been but scantily handled by Dr. Hope and Dr. Walshe. The former merely states, that the signs, as far as he can judge, are:—1st. Diminution of the sounds, especially the first. 2nd. Irregular pulse, without valvular disease. 3rd. Oppression, or even pain in the præcordial region, with general signs of a retarded circulation, producing cerebral, hepatic, and pulmonary congestions. These signs, taken in conjunction, are, he observes, peculiar, because, while No. 1 is proper to simple hypertrophy, Nos. 2 and 3 are foreign to its early stages. The aggregate, therefore, probably denotes an incumbrance of the organ with fat."[1]

Dr. Walshe observes, that "the physical signs are those of a soft heart; weak impulse; indistinctness of the apex beat; unchanged percussion dulness (unless there be alteration of bulk from some other cause;) a feeble, toneless, short, first sound; a long first silence; and a feeble second sound (this may be of better tone at the second left than the second right cartilage, if the fatty degeneration be, as it often is, in great excess in the left ventricle.) Possibly a dynamic mitral regurgitant murmur may sometimes occur, but I do not know this from observation. The pulse is irregular in force and rhythm, either constantly or from time to time, under excitement, the influence of flatulence, indigestion, effort, &c. On such occasions it may become exceedingly frequent. I have known it uncountable, in the main from frequency, in part, however, from irregularity. Infrequency of pulse, occasionally met with, is in some cases referrible to the weakness of occasional systoles, but the systoles are themselves sometimes much less frequent than natural."[2]

Dr. Walshe shortly describes the general symptoms; but with the exception of the tendency to syncope, there is nothing special in them, nothing different from those met with in many diseases of the heart. And this observation applies to the more extended analysis of the general symptoms which has been given by Dr. Quain. The important characteristics of the disease in its confirmed state were described by Dr. Cheyne and Dr. Adams; and the cases long ago published by these observers, and also those by Dr. Townsend and Dr. Law, furnish examples of the peculiar slow pulse, the pseudo-apoplectic attacks, and, in fact, of all the great features of the disease.

The results of my investigations into the state of the heart in typhus fever, published in 1834, especially those which refer to the physical signs, had an important bearing on the diagnosis of fatty degeneration of the heart, at least of that form where the action of the heart is regular. In both we may observe the feebleness, and sometimes the extinction, of the systolic sound, and the want of impulse. Under certain circumstances, a slow action of the heart is met with in the convalescence from typhus. Thus, the phenomena of the two diseases become mutually illustrative, although their natures are different.

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[1] "A Treatise on Diseases of the Heart," &c., p. 324. Fourth Edition, 1849.

[2] "A Practical Treatise on Diseases of the Lungs and Heart," p. 494.

ON THE ARCUS SENILIS AS AN ATTENDANT ON FATTY DISEASE OF THE HEART.

As I have no original observations to record on this subject, I shall content myself with referring to Mr. Canton's interesting paper, in which he shows that the arcus senilis is caused by fatty degeneration of the cornea. Dr. Williams has found that the appearance in question is commonly met with in persons who present symptoms of weakened heart; and Dr. Quain alludes to a case in which this morbid state of the cornea was well marked, and the heart was in the fatty condition. He observes, that when the signs and symptoms of fatty degeneration of the heart are present, this appearance of the eye will aid in the diagnosis. Mr. Canton has found the degenerated state of the heart in cases of well-marked arcus senilis.[1]

OCCURRENCE OF AIR IN THE HEART, VEINS, AND SOLID VISCERA, SOON AFTER DEATH.

The observation of an unusual appearance of congestion in the superficial veins soon after death, as well as that of the production of air in the heart and viscera of the abdomen in this disease, is due to Professor Smith. Within a short period after death he has found the heart inflated like a balloon, so that on raising the sternum it burst out from the chest. This was before the commencement of putrefaction. The veins, too, contained air in quantities, and along the course of the superficial vessels he observed a peculiar mottled appearance of the surface. The liver, spleen, and kidneys may be found so full of air as to float upon water; and the general aspect of the body is such, that in many instances he foretold with accuracy that the heart would be found in the fatty state, even though he had not seen the patient during life.

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[1] Op. cit. p. 161.

This rapid production of air seems connected with the oily state of the blood, and the existence of air-bubbles in the veins is to be noted as showing that the air proceeds from the decomposition of the blood rather than of fatty matter in the organs themselves. In a case of vast abdominal tumour which occurred some years ago in the Whitworth Hospital, the epigastric veins, which were varicose and tortuous, were found to contain air in considerable quantities, for several days before death. Large bubbles of air could be easily felt in them, and might be pressed along by the finger. In this case the blood was in all probability in the oily state. Under these circumstances, emphysema of the internal organs occurs before the ordinary appearances of putrefaction have been established.

I once saw it in the liver of a man who died from the rupture of a small aneurism of the aorta into the œsophagus, where for a length of time it had so pressed as to prevent deglutition. The organ was of a yellowish white colour, and evidently in a state of fatty degeneration. It floated on the surface of water, and so great was the development of air that many portions of it, and the whole of the lobe of Spigel, could only be compared to emphysematous lung. In this case the condition of the blood was probably much depraved, for so complete was the obstruction of the œsophagus, that the patient was dying of inanition when the fatal rupture took place.

In Dr. Graves's Clinical Medicine there are some interesting observations on the occurrence of emphysema, supposed to arise from great losses of blood. He quotes from a memoir by M. Rérolle de Gex, who has given two cases of emphysema. In one, œdema occurred after profuse epistaxis, and the patient sank rapidly. The veins contained a great quantity of air, and in both auricles coagula, in an emphysematous state, were found. No air was discovered in the arteries. In his second case, profuse hemorrhage had followed upon the operation for the removal of a large tumour, and on the following day the extremities were emphysematous. In this case, as in the former, a long and alarming syncope followed the loss of blood. On dissection, the heart was found collapsed; there were coagula in the right cavities containing air. The smaller veins were filled with air, and when portions of the muscles were squeezed before a candle, jets of an inflammable substance were produced, similar to those seen when we compress an orange-peel under the same circumstances. It was also found that an inflammable gas was emitted from every portion of the cellular membrane when divided by a bistoury. This gas was inodorous, and burned with a slight detonation, and with a bluish-white flame.

Dr. Graves notices a case in which excitement of the heart, with a thrilling pulse, ended in repeated attacks of profuse epistaxis, causing extreme debility, and followed by emphysema of the subcutaneous cellular membrane of the abdomen. It is more than probable that in some of these cases, as well as in others of spontaneous emphysema recorded by various authors, an oily state of the blood really existed; and the cases may also have been examples of fatty disease of the heart.

The development not only of inflammable air in the cellular membrane, but of a substance, also inflammable, which was spirted forth from the muscular structure on pressure, makes it important that the investigations lately undertaken by Bischoff and Liebig, as to the possibility of spontaneous combustion, should be extended to cases of fatty degeneration of the heart and other organs.[1]

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[1] See Dr. Graves's Clinical Medicine,—Article Emphysema after Profuse Hemorrhage. The facts from the thesis of M. Rérolle de Gex are given in the "Gazette Medicale de Paris," tom. iii. No. 103. The case by Bally, quoted by Professor Apjohn in his Treatise on Spontaneous Combustion, in the Cyclopædia of Practical Medicine, should be consulted; as also the investigation concerning the death of the Countess Goerlitz, Edinburgh Medical and Surgical Journal, No. cxcii., in which are given the views of Bischoff and Liebig, as opposed to the doctrine of spontaneous combustion.

In the case by Bally, as in that by Rérolle de Gex, it was found that a gas escaped on cutting into any emphysematous part, which ignited at a candle, and burned with a bluish flame. Professor Apjohn admits that a spontaneous though limited decomposition may take place in the living body, attendant on the diminution of the powers of life, which is the consequence of disease, so that new arrangements of the elements of organized matter may be produced, similar to those which commonly follow the total extinction of the vital powers by death. It is not to be forgotten that, in the alleged cases of spontaneous combustion, as is remarked by Professor Apjohn, the calamity seems peculiar to the old and feeble, to the corpulent, to individuals greatly emaciated, to inactive persons, and to those addicted to the abuse of spirituous liquors.

CHAPTER VI.

TREATMENT OF THE ORGANIC DISEASES OF THE HEART.

It has been too much the practice of medical writers, when they have to deal with the diseases of an organ such as the heart, to discuss the treatment of each of its affections separately. The objection to this method is, that it assumes too strongly that each disease is presented in an isolated form. For example, we find rules laid down for the treatment of hypertrophy of the heart; of dilatation as distinguished from hypertrophy; of diseases of the left as differing from those of the right side of the heart; as well as of the various forms of disease of its orifices. It is undeniable that many of these precepts are founded in truth, and that, if we only considered the mechanical state of the heart, we might adopt much of what has been written as to the treatment of its isolated diseases. Thus the treatment of active hypertrophy will differ from that of passive dilatation; the treatment of contraction of the mitral orifice from that of permanent patency of the aortic valves; and again, fatty degeneration will demand a course differing from that required when the contractile force of the heart is increased.

The objection to this mode of studying the subject is, that it not only assumes the isolation of diseased conditions, but overlooks the great fact, that in many of these cases, changes, not only in the mechanical but the vital state of the organ, are continually going on; and that, even with the existence of organic disease, the state of the blood has a great influence on the physical signs, and also on the results of treatment. Nor is it to be forgotten that the most prominent of these signs do not always belong to the original and more important disease, which lies, as it were, hidden by the effects of the disturbance which it has itself excited.

But these considerations are in nowise intended to discourage the study of treatment in special diseases of the heart. A time may come when the science of diagnosis will be carried to such perfection that we shall minutely determine not only the condition of each portion of the heart, but discover the rise and watch the progress of every molecular change in its structure, and every mutation of its vitality. At present we must be content with such knowledge as we possess, and above all things, avoid injuring the cause of practical medicine by laying down rules of treatment founded upon insufficient data. Let the practitioner keep this truth steadily before him, so often insisted on in this work—that the vital rather than the mechanical condition of the heart is to be his great guide in practice. Let him have a competent knowledge of the effects of disease of the heart upon other organs, and of its own sympathetic derangements, and he will scarcely err in the treatment of cardiac affections.

It is objected to the pathological science of the present day, that it has been too much occupied with the study of the nature and diagnosis of disease, and too little with its treatment; and the works of the continental physicians are especially pointed out as being fruitful in facts of pathological anatomy, and barren in those of practical medicine. But more has been said on this subject than is, perhaps, just; and we should be careful not to decry the labours of men who have done so much, because they have not effected everything. For medicine is a progressive science, and the pathological anatomist is truly the pioneer of the physician; and the latter, if he desires to advance his science, must train his mind to a higher exercise and a more comprehensive thought than that required for the observation and recording of isolated facts in pathology. He must not only give due weight to the existence of separate phenomena, and to the results of combinations of diseases, but he must study conditions which are beyond the reach of the pathological anatomist, and learn to ignore the existence even of manifest organic alterations. While he bears in remembrance the ascertained facts supplied by modern investigation, he is to go beyond them, having an eye to the general rather than the particular, and to found his diagnosis, prognosis, and treatment on that broad basis which includes not alone the effects, but the causes and special modifications of disease. Thus only can the new medicine be advantageously combined with the old. Thus only can that kind of skill which is, as it were, the instinct of the thinking man, be produced—the true *mens medica*—enabling us to act rapidly and successfully in opinion and practice, unconscious, too, of any effort of memory or complicated reasoning process. This is the great desideratum in medicine, when we are called on to deal with varying combinations of local diseases, and general morbid conditions. Accurate observation and accumulated experience are necessary for its attainment; and yet it is more accessible to some than to others.

A few, indeed, seem incapable of acquiring it; and hence it is that, with a more limited experience, the mind of one man shows more brightly than that of another. In medicine we have to deal with ever-varying phenomena; the vital condition of organs cannot always be inferred from their physical state, nor the influences which act on the entire economy be explained by anatomy. Were it otherwise, medicine would be an easy pursuit, but no field for this exercise of mind, which is at once its difficulty and its glory.

In this extra-anatomical study of disease the effect of treatment is a first element. Dr. Latham well observes, that—"The treatment of diseases, rightly considered, is, in fact, a part of their pathology. What they need and what they can bear, the kind and strength of the remedy, and the changes which follow its application, are among the surest tests of their nature and tendency."[1]

Having already examined into the treatment of pericarditis and endocarditis, we may now consider that of the ordinary chronic affections of the heart, especially of hypertrophy, dilatation, valvular disease of the left side, and the cases of diminished power of the heart, connected with fatty degeneration.

TREATMENT OF HYPERTROPHY OF THE HEART.

In the treatment of this affection we must not forget that cases are met with in which the force of the heart is but little if at all increased; and, again, that we meet examples of very large and thickened hearts in which the symptoms are only to be moderated by the use of stimulants. The mere existence, then, of physical signs and symptoms of hypertrophy, even if they are really indicative of the disease, should not necessarily cause the physician to debar the use of stimulants, or to adopt a lowering system of regimen or medicine. It is often in its influence on the secondary symptoms rather than on the apparent condition of the heart or pulse, that we observe the effect of the lowering system in these cases. Thus examples may be met with in which the orthopnœa is always rendered more distressing when stimulants are removed.

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[1] Lectures on Subjects connected with Clinical Medicine, &c. By P. M. Latham, M. D. London: 1845.

To such instances we might give the name of passive hypertrophy, in contradistinction to that class in which excitement of the organ is found. Doubtless, it is in cases of hypertrophy with permanently patent aortic valves that we most frequently observe the necessity for stimulation, and the danger of an antiphlogistic treatment; but the same may occur in hypertrophy independent of disease of the valves.

The indication of treatment in the more active forms of the disease is to moderate the force and general excitement of the heart's action; for we must agree with Dr. Latham in the opinion he has so strongly expressed as to the incurability of confirmed hypertrophy—not that there is any reason why such a result should be impossible, but that evidence is still wanting to prove that a cure of true hypertrophy has ever been made; and this being the case, the attempt to remove a probably incurable affection, by means which will depress and injure the general health, must be discouraged.

It is observed by Hope, that the art of treating hypertrophy consists in keeping the patient rather low, and the circulation very tranquil, yet short of producing anæmia or debility. When this desirable state of calmness without debility can be obtained, the heart, according to this author, possesses a surprising power of reverting to its natural size; and this, he observes, ought not to be wondered at when we reflect on the rapidity with which the muscles of voluntary motion, even when hypertrophied, as in the cases of dancers, smiths, &c., will emaciate and become feeble when their exercise is wholly suspended.

But when we recollect that hypertrophy of the heart, as an uncomplicated disease, is rare, and that even if the pericardium, valves, and aorta were healthy, yet that the very existence of hypertrophy may indicate a bad state of the general health,—we must be slow to adopt any course that would weaken the heart, or depress the system at large. The indications of treatment are in theory sufficiently plain; but who can tell whether the process of reduction of the volume of the heart, once set up, can be made to stop exactly at the point when the organ is restored to its natural dimensions? And who can say that in this very attempt to cure one disease, we may not predispose the patient to another and a totally different affection? The question involves the whole subject of muscular atrophy. If the observations of Retzius, on the reduction of volume of the puerperal uterus, be found applicable to cases of atrophy of either the involuntary or voluntary muscles, it might be found that in reducing the volume of the heart, we run the risk of setting up one of its worst diseases.[1]

When, therefore, the practitioner has to decide whether he will attempt the curative, or content himself with the palliative treatment of hypertrophy of the heart, he must duly weigh the following points:—

1. That hypertrophy of the heart, occurring independently of disease in the valves or the aorta, is the exceptional case.

2. That disease of these structures may be progressing, and yet without any physical sign to indicate its presence.

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[1] The observations of Retzius, on the diminution of the uterus after childbirth, appeared in the Hygieia of Sweden in 1851, and are alluded to by Professor Simpson in his "Contributions to Obstetric Pathology and Practice," in the Monthly Journal of Medical Science, August, 1852. Professor Simpson well remarks, that "if the process of absorption in organs is ever studied successfully anywhere, it will probably be by making observations on the reduction or involution of the uterus in women or in the lower animals, subsequent to parturition." And this observation is justified by the discovery of Retzius, not only that the absorption of the walls of the puerperal uterus is preceded by fatty transformation of their muscular fibres, but that the blood, during the convalescence of the puerperal woman, shows under the microscope a superabundance of fat globules. How strongly this last fact bears on the views of Professor Smith as to the origin of the fatty heart need hardly be pointed out. I do not wish, however, to be understood as adopting the opinion, that every case of muscular atrophy implies as a first, or at least an early step, the occurrence of fatty degeneration,—for the rapid emaciation of a single limb, as in cases of painters' paralysis, and also as a premonitory sign in chronic disease of the articulations,—a fact long ago taught by the late Dr. Colles,—makes it appear that in such cases the fatty change, if it occurs, is consequent on the atrophy.

3. That in the complication with affections of the aortic valve, both theory and experience are opposed to the use of an antiphlogistic treatment.

4. That the liability to the anæmic state in these patients should make us watchful how we reduce the general strength, or lessen the mass of the blood.

5. That the anæmic condition may, on the one hand, cause great excitement of the organ, and, on the other, predispose to its fatty degeneration.

The treatment pointed out by many writers consists in the use of general and local bleeding, setons, issues, and the employment of various sedatives.

The use of the lancet should rarely be adopted, and the practice of repeated venesection in this disease cannot be too strongly reprehended. On this point Dr. Latham has insisted with great force, dwelling on the effects of the anæmic state when superadded in a case of hypertrophy.[1]

The same objections do not apply to the use of local bleeding, and in many cases we may, by applying small numbers of leeches from time to time, effect a great improvement in the action of the heart. But even this treatment should not be pushed too far. Cupping is often objectionable from the excitement which is induced at the time of the operation.

With respect to the use of issues and setons, it may be said that their efficacy is by no means established. Of the two methods, the issue is to be preferred. Indeed, the insertion of a seton over a heart acting with violence is sometimes attended with a hæmorrhage which may be very difficult to control. I have known some most troublesome and unhappy cases of this kind; and to both these modes of treatment there is the objection, that they act in causing the attention of the patient to be continually directed to his heart, than which there is nothing more likely to keep up or augment its morbid activity. It may be that their employment would be more justifiable in cases where the signs of hypertrophy are recent, and following on an attack of endo-pericarditis.

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[1] "Beware, then," says Dr. Latham, "in the management of hypertrophy of the heart,—beware, above all things, of bleeding your patients into paleness and poverty of blood."—*Lect.* xxxi. The indisposition to use the lancet in chronic diseases, and in a vast number of acute affections, especially in fever, is one of the best evidences we can have of a safe and scientific practitioner.

The application of small blisters is sometimes advantageous; it appears better to repeat them than to expose the patient to the annoyance of an open suppurating surface. In a few instances I have tried the endermic employment of digitalis, so eulogized by Bouillaud, but without any distinct result.[1]

The method of Bouillaud consists in the application of a blister to the præcordial region, and then sprinkling the vesicated surface every day with from six to fifteen grains of the powder of digitalis. "Thus," he says, "as by enchantment, we may diminish the number and force of the pulsations of the heart." And it is plain that, should experience confirm the efficacy of this treatment, it has advantages over the internal exhibition of the remedy, which is so apt to cause a weakened state of the stomach.

Of the sedatives which have been employed in the disease of active hypertrophy of the heart, the most generally useful are the different preparations of digitalis and the hydrocyanic acid. Dr. Hope speaks highly of the extract of aconite, especially in cases where there is pain, but I have no experience in the use of the remedy. With respect to the exhibition of digitalis, it appears to answer best when given in such doses as that its use can be continued for a length of time; and the choice of the particular preparation of the remedy may be left to the discretion of the practitioner. The tincture or the infusion will generally be found the most advisable.

The treatment I have found most useful in those cases which may be described as simple hypertrophy of the heart consists in the application of small numbers of leeches to the præcordial region, at intervals varying from three to six days, while at the same time the patient is made to take from six to twelve drops of the tincture of digitalis three times in the twenty-four hours. This treatment is to be continued for some weeks, provided that the depressing effects of the remedies do not exceed the bounds of safety. Should the digitalis produce nausea or loss of appetite, we may substitute the medicinal hydrocyanic acid in doses of from one to two drops three or four times a day. Its best vehicle will be distilled water. We may use anodyne plasters over the region of the heart; or, should these be objected to, we can apply the extract of belladonna. In this way the violence of the action of the organ may be greatly reduced, and in a few cases permanently removed. The good effects of the endermic employment of belladonna should make us study a similar mode of using other sedatives. The treatment of Bouillaud has been already alluded to, but the entire subject of the endermic use of sedative medicines in the active diseases of the heart, both inflammatory and non-inflammatory, requires investigation. Indeed, when the effect of belladonna or of atropine on the iris is remembered, we might expect benefit from the remedy in lessening the action of other involuntary muscles.

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[1] The observations which I have made on the endermic use of digitalis are neither sufficiently numerous nor accurate to enable me to pronounce either for or against this mode of treatment. If we adopt the method of Bouillaud, care should be taken that the digitalis is good, and in the finest possible state of division. It is still to be determined how far we may employ Digitaline in this way; for although the researches of Homolle and Quevenne have shown that the very small quantity of a milligramme (about the sixty-fifth part of a grain,) when applied to a surface denuded of epidermis, excited inflammation and painful tension of the surrounding parts, yet it is possible that it might answer in the form of an ointment applied to a healthy surface. ("Mémoires sur la Digitaline, par MM. Homolle et Quevenne. Rapports faits a l'Académie Nationale de Médecine." Paris, 1851.)

When the tongue is clean, and febrile excitement absent, the internal use of digitalis is of the utmost benefit in active hypertrophy of the heart.

The following are the conditions in which we may expect most advantage from the treatment now indicated:—

1. The disease occurring in a young or middle-aged person of a good habit of body.
2. A healthy state of the digestive function, as shown by the cleanness of the tongue, the colour of the skin, and the condition of the urine.
3. Absence of fever of any kind.
4. Absence of complication with the signs of valvular disease, especially of those which indicate regurgitation through the aortic valves.

On the inutility of the hydriodate of potass, given with the view of diminishing the volume of the heart, my experience coincides with that of Dr. Hope. I have never employed this treatment, but have known cases in which it was attempted without any good result.

Among the palliative remedies we must place narcotics in the first class; especially in cases where the increased action is attended with pain,—opium, hyosciamus, hydrocyanic acid, the Indian hemp, and chloroform, may all be exhibited. In some instances, as in the case of vast hypertrophy by Dr. Croker King, the patient becomes inured to the action of narcotics, and requires large quantities to produce any soothing effect.

TREATMENT OF HYPERTROPHY OF THE HEART IN CONNEXION WITH VALVULAR DISEASE.

It is to Dr. Corrigan that we owe the establishment of true principles in this part of practical medicine. For although his researches, as well as his observations on treatment, have reference to the combination of hypertrophy of the heart with imperfection of the aortic valves, yet every practical man must see that those principles, so ably indicated, may be extended to cases in which the mitral orifice is the seat of the lesion. Great injury is inflicted on both classes of patients from neglect of Dr. Corrigan's rules of treatment; for we every day meet with cases severely damaged from the double error having been committed, —first, of taking a long-existing and incurable affection for a recent disease, and next, of using an antiphlogistic treatment, the effect of which is to counteract and nullify the anatomical arrangements which nature has made for the preservation of life.[1]

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[1] "The disease," says Dr. Corrigan, "we are considering, is an inadequacy in the valvular apparatus at the mouth of the aorta permitting a regurgitation of blood into the ventricle. In the perfect state of the valvular apparatus at the mouth of the aorta, the valves support by intervals the column of blood in the aorta, and the heart, with its ordinary complement of fibre and of muscular strength, is with this assistance competent to the office it has to perform. But when, in consequence of a deficiency in the valvular apparatus, the heart does not receive its due share of assistance from these valves, and is obliged to perform not only its own function of propelling the blood, but has in addition to support after each contraction a portion of that weight of blood which should then be wholly supported by the valves, it is no longer in its ordinary state equal to the task imposed upon it. In such circumstances, nature, to enable the heart to perform the additional labour thrown on it, increases its strength by an addition of muscular fibre, and the heart thus becomes hypertrophied, in accordance

It is certain, too, that the evil effects of a reducing treatment in these cases are partly attributable to the fact that the heart is often in a state of commencing fatty degeneration; so that whether we look to the effect of antiphlogistic treatment as

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with the general law, that muscular fibre becomes thickened and strengthened when there is additional power required from it. Is this hypertrophy disease, or is it a wise provision of nature, by which the organ is thus made equal to the increased labour it has to perform? On the answer depends the treatment to be adopted; and on this there is no room for hesitation. A heart of ordinary strength could not, under the circumstances, carry on the circulation; and nature then wisely endows the heart with the requisite degree of strength. It is at once obvious, that to interfere with this wise provision of nature, to diminish the strength of the heart, or, if we choose other words, to direct, according to the advice of Laennec, Bertin, &c., our measures against the hypertrophy of the organ, is to deprive the system of the only power which enables the heart to carry on the circulation. No one thinks of directing measures to diminish hypertrophy of the muscular tissue of the stomach in narrowing of the pylorus, or from scirrhus of the bladder or rectum in stricture of the urethra or intestines.

"In these instances the hypertrophy is recognised as a provision of nature to make the power of the part equal to the obstacle it has to overcome; and yet this simple principle seems to have been entirely overlooked in diseases of the heart, as if this organ possessed muscular fibres of a different nature from other organs, or as if, in adapting itself to obstacles affecting its action, it follows laws different from other muscular parts. The consequence of the neglect of this principle has been, that too often, in treatment of a valvular alteration in the heart, there has been a constant struggle between nature and medicine. Nature has been making the organ equal to its task: while medicine has been directed to counteract nature's efforts, and, by weakening the organ, to render it totally incapable of its task. The repeated bleedings, the starvings, the enforcement of debilitating measures, are totally unsuited to the disease we are considering.

"Instead of such treatment, the measures most beneficial are those which, by strengthening the general constitution, will give a proportionate degree of vigour to the muscular power of the heart, and thus enable it to carry on the circulation in the absence of that assistance which it ought to receive. With this view, a generous and sufficient diet of animal and vegetable food should be advised, at the same time that an abstinence from those beverages, such as malt liquors, which increase much the mass of the fluids, should be enjoined. It is not at all necessary that the patient should be prohibited from attending to his business or profession, provided that he do not devote to it so much attention as to produce debility. And as there is among patients who have learned that they are afflicted with heart disease, a universal dread of sudden death, it is necessary to undeceive them on this point; and in the present instance it can be done with perfect safety, as the termination of the disease is never sudden. This plan of treatment, opposite to what has been generally enjoined, was forced upon the attention long before the reasoning adduced here had been brought to support it."—See Dr. Corrigan's Memoir, *Edinburgh Medical and Surgical Journal*, vol. xxxvii. 1832.

simply diminishing the power of the heart, supposing it free from degeneration, or as acting on an organ already enfeebled by interstitial change, we must admit, even if experience did not declare the fact, that this sort of interference is mischievous in a high degree. Of the truth of these principles, the following is a good example.

A man who had long laboured under the disease, yet without any important suffering, consulted a practitioner, who at once applied a large number of leeches over the heart. The effect was remarkable and immediate, for dreadful paroxysms of cardiac asthma, succeeding each other at short intervals, were for the first time established. I treated this patient with the aromatic mixture of iron, in conjunction with nourishing food and small quantities of wine, and after some time succeeded in restoring him to his former condition of health, which was quite sufficient to enable him to follow the business of head waiter at a hotel.

In these cases a condition indicated by Dr. Corrigan may arise, which I myself have not met with,—it is that of an extraordinary increase in the action of the heart, and in the strength of the pulse, and yet there is no fever or local inflammation to be detected. The patient suffers from straitness of the chest and difficulty of breathing, and the symptoms appear to arise from an increase of bulk in the mass of circulating blood, which keeps the vessels so distended that the heart becomes distressed, incapable of freely contracting, and tumultuous in its action. Relief is at once afforded by a large blood-letting, followed by an opiate. Dr. Corrigan properly observes, that the employment of a full bleeding in these circumstances is very different from the repetition of those irritating small bleedings which are commonly practised.

When inflammatory attacks supervene in this class of patients, I believe that in the employment of the lancet we must exercise great caution. To this point I have already alluded. Where acute inflammation, such as in pneumonia, peritonitis, or rheumatism, occurs, Dr. Corrigan advises a prompt, but not protracted, use of the antiphlogistic treatment, and he observes as a curious fact, that bleeding carried to a large amount does not produce fainting. He advises a free bleeding, but that when the inflammatory affection is once subdued, we should cease as soon as possible from debilitating treatment, which, if persevered in, would prove injurious to the organic affection.

I cannot offer any results of my experience on this question, having always avoided the use of the lancet in these cases.

TREATMENT OF THE WEAK AND PROBABLY DILATED HEART IN CONNEXION WITH ENLARGEMENT OF THE LIVER AND PULMONARY DISEASE.

Having now considered the treatment in those cases where the muscular contractility of the heart is increased, in connexion with actual hypertrophy, we next proceed to examine that of the weakened hearts, of which the two most common forms are dilatation of the cavities, associated with pulmonary and hepatic disease; and again, the fatty degeneration.

The general principles of treatment in both these cases are the same. We should avoid all reducing measures; we must endeavour to improve the condition of the blood, and, by stimulants and tonics, increase the power of the weakened and atrophied muscular fibres. And the practitioner should bear in mind, that he has to deal with a very complicated case, and that the heart affection may be but the manifest exponent of a diseased condition of the entire system. But although the great principles of treatment are the same in the weakened and dilated heart, such as occurred in the case of Dr. Colles, and in either form of the fatty heart, yet in one important particular we find them to differ. I allude to the beneficial action of mercury in relieving many of the symptoms, and removing the consequences of the first of these diseases.

It is remarkable, that although the beneficial action of mercury in this affection is known to many practical physicians, but little information can be found on the subject in any of our medical works; and yet it is impossible to speak too highly of the efficacy of this treatment in a certain class of cases. I do not wish it to be believed that by mercury we can cure dilatation of the heart; but many years' experience has convinced me that by the use of this remedy we can delay its production, remove the irregular action which assists in causing the disease, and, above all, prolong the patient's life, and again and again relieve him from dropsy, and from pulmonary and hepatic congestions, even when they have arrived at a point which threatens a speedy dissolution.

I have already, in speaking of the case of Dr. Colles, alluded to the reciprocal action which exists between the liver and the heart, and shown that it is often impossible to declare whether the affection of the heart or the liver has had the initiative. When what may be termed the paroxysm of the disease is established, we find that in one case the exciting cause has primarily acted on the heart, and in another on the liver,—but which of these cases is the more common we are not in a position to state. That there is a morbid state of the liver, capable of producing great irregularity of the heart's action, is certain; yet we find, so far as the heart is concerned, these patients presenting very opposite conditions. Thus the action of the heart may be excited, rapid, and irregular in the last degree, and so continue for months together, when either spontaneously, as it were, or after treatment directed to the digestive system, its motions and sound become perfectly natural, and there is not the slightest indication of change in the volume or capacity of the organ. I do not know any case more full of instruction than this, for it happens that opposite opinions are often entertained as to the state of the heart by well-informed physicians,—some declaring the organ to be perfectly healthy, others, that it is in the very last stage and extreme of disease. The discrepancy arises from this, that one observer sees the patient in the period of tranquillity of the heart,—the other, during its extraordinary excitement. That the frequent returns of long periods of irregularity of the heart would ultimately produce organic disease can scarcely be doubted; yet I have lately seen a case of several years' standing in which the invalid presented the two distinct conditions above specified, and in which, after mercurial action, the heart became so perfectly regular, and its phenomena so natural, that it was difficult to persuade one's self that the patient was the same person who had been examined but a few days before.

In this case there was a permanent hepatic tumour, and during the violence of the cardiac symptoms the liver was extremely tender on pressure. The patient, however, was not jaundiced, nor did he present symptoms of inflammatory fever.

This case may serve as an introduction to the more common examples of weakened hearts in which so much benefit is derived from mercurial action. In such cases the following circumstances are to be met with:—

1. The patient is generally advanced in life,—most of the cases being in persons of from 50 to 70 years of age.

2. These individuals are originally of healthy constitution and strong habit of body.

3. They are liable to some degree of gout, which malady, after having long occurred in its more regular form, becomes masked or imperfect.

4. They are subject to bronchitis, which, during the aggravation of the symptoms, increases so as to resemble suffocative catarrh.

5. The liver is permanently enlarged, yet in many no appearance of jaundice exists. The hepatic tumour is generally indolent, and the epigastric veins are seldom varicose.

6. Two conditions of the heart may be observed. In both there is *permanent* irregularity, always augmented during the paroxysm of suffering; but in one class of patients the physical signs indicate hypertrophy with valvular disease, often affecting both orifices of the left ventricle; while in the other the signs are those which have been indicated in the chapter on Dilatation of the Heart, unattended by any direct indication of valvular disease. These patients are from time to time liable to the occurrence of dropsy, which, commencing in the extremities, will, if not soon checked, invade the entire system. This condition is preceded by diminished action of the kidneys; the heart becomes more excited and irregular; the liver swells like an erectile tumour, and the lungs are oppressed by congestion. Orthopnœa is established, attended with the worst paroxysms of cardiac asthma. Ascites may appear, and the patient's life be threatened daily. Yet, under all these terrible symptoms, it happens again and again that the exhibition of mercury will, as by enchantment, remove the anasarca, reduce the hepatic tumour, restore the heart to its ordinary, though not its normal condition, and for a period of time, more or less

long, enable the patient to pursue the avocations of an active and laborious life.

The quantity of the remedy which is required, as we might expect, varies in different cases. In some it is requisite to establish ptyalism, while in others the relief of the heart, and the disappearance of the dropsy, are observed after the use of a very mild course, in which little if any of the characteristic action of mercury can be perceived, unless we include diuresis. In other cases it will be necessary to use diuretics following on the mercurial action, and in this way we often observe a singularly abundant secretion of urine, attended by rapid subsidence of the dropsy and visceral oppression. We should use various combinations of the vegetable and saline diuretics; and even digitalis, in connexion with diuretics of the tonic and stimulating class, may be employed. The success of diuretics appears to turn upon their being preceded by mercury. I have often, in cases where the patients for former attacks had already used a great deal of mercury, attempted to remove the dropsy by diuretics alone, but have always failed, and yet found that a diuretic which, without the previous administration of mercury, was totally inefficacious, acted vigorously when given after a few days' use of that remedy.

But the truth is, that in these cases we are not to be over timid in the repetition of mercurial medicines; for there is nothing more remarkable than the power which the patients exhibit of bearing repeated courses of mercury not only without injury, but with extraordinary benefit to their general health. In some, indeed, the state of aggravation of symptoms appears to be kept off for an indefinite period by the continued use of small quantities of the medicine. The patients will improve in flesh, appetite, strength, and appearance. In others, as in a remarkable case which I have lately seen, the repeated use of very slight courses of mercury, at short intervals of time, has preserved the life of the patient for several years, and enabled him to pursue a laborious profession. This gentleman has now had not less than thirty distinct courses of mercury. It is truly his "pabulum vitæ;" and neither in this case, nor in any of the others in which I have seen the treatment pursued, were the injurious effects of mercury ever produced. There has been no unhealthy action on the mouth,—no periostitis, cutaneous eruptions, or tremors.

It need hardly be observed, that a time at last arrives when, as in the case of Dr. Colles, the system no longer responds to the action of medicine, and the patient sinks with dropsy and pulmonary congestion.

During this treatment, and especially when free diuresis is established, it is necessary that wine or some other diffusible stimulus should be carefully administered, and the system supported by a proper aliment; for there is nothing more dangerous than by any interference with the usual habits of the patient to reduce the strength in these cases.

TREATMENT OF FATTY DEGENERATION OF THE HEART.

We cannot yet say whether a heart once in the state of fatty degeneration can ever be restored to health, yet it does not seem impossible that such a result might be obtained if we had to deal with an uncomplicated case, and could recognise the disease at an early period. But as, in most instances, this change occurs in advanced life, and is combined with atheromatous disease of the aorta, or with bronchial or hepatic affections, or further associated with the gouty, strumous, or anæmic conditions, the chances in any given case are greatly against the restoration of the organ to health. On the other hand, analogy would seem to indicate that even after atrophy of the heart has been produced, both its force and volume might be restored. Such a result would be more likely to be produced in cases where the adipose substance is first deposited externally, pushing the muscular mass before it, and dipping into its interstices. We must be cautious in too narrowly limiting the powers of nature; and as it is known to practical physicians that, by the adoption of proper measures, we can often remove the early symptoms of debility of the heart, there seems no reason to doubt that with the advance of medicine, both diagnostic and hygienic, we may yet be enabled not only to check the growth of fat in the heart, but to restore the muscular fibre to its pristine condition of volume and of power.[1]

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[1] The views of Dr. Quain, as to the chemical nature of the fatty transformation of the heart, might be appealed to as opposed to the doctrine of the possible curability of this disease and the restoration of the muscular fibre of the heart (see page 335.) But even if the analogy between the alteration of a dead portion

In the present state of our knowledge, the adoption of the following principles in the management of a case of incipient fatty disease seems justifiable:—

1. We must train the patient gradually but steadily to the giving up of all luxurious habits. He must adopt early hours, and pursue a system of graduated muscular exercise; and it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise with pleasure and advantage, which at first was totally impossible, owing to the difficulty of breathing which followed exertion. This treatment by muscular exercise is obviously more proper in younger persons than in those advanced in life. The symptoms of debility of the heart are often removable by a regulated course of gymnastics, or by pedestrian exercise, even in mountainous countries such as Switzerland, or the highlands of Scotland or Ireland. We may often observe in such persons the occurrence of what is commonly known as "getting the second wind," that is to say, during the first period of the day the patient suffers from dyspnœa and palpitation to an extreme degree, but by persevering, without over-exertion, or after a short rest, he can finish his day's work, and even ascend high mountains with facility.[1] In those advanced in

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of muscular fibre, placed in contact with chemical re-agents, and the condition of the muscles of the heart in this disease, was stronger than it really is, we should not on that account despair of arresting or changing the morbid process in the heart. The vital power of the organ, though diminished, is still there; and it would be a bold thing to say that even the fat globules themselves are not under the influence of the general vital force. It is a dangerous matter to discourage attempts at the cure of a disease by declaring the imposibility of such an occurrence; for under these circumstances, the less scientific morbid anatomist, though often the more practical physician, will be disheartened, and in despair give up any attempt to cure that which has been declared incurable. The whole history of specificism in medicine seems to point out that the healing art will yet greatly advance in this direction; and who can say that, in addition to the hygienic measures and medical treatment properly pointed out by Dr. Quain, and before him, by Latham, Hope, and other authors, as seen in their writings, not only on this affection, but generally on weakness and dilatation of the heart, we may yet discover measures by which the formation of fat globules may be arrested, or even the local development of muscular structure increased.

[1] It appears likely that the effect of pedestrian exercise is twofold, and that it not only increases muscular energy and development, but also, by augmenting the secretion from the skin, assists in carrying off a large quantity of the oily consti-

life, however, as has been remarked, the frequent complication with atheromatous disease of the aorta and affections of the liver and lungs must make us more cautious in recommending the course now specified.

2. We should advise the use of such a regimen as will tend to nourish without increasing the bulk of the system, and especially the growth of fat. The patient may use fresh meat of any kind freely, but should avoid taking an over-quantity at a particular meal. He should abstain from all articles of food which are oleaginous, and probably also the white meats. He must be forbidden the use of soup or of much milk, and should partake of vegetables sparingly. His use of fluids should also be as sparing as possible. The best drink would be water, with or without a little brandy or wine; but soda water, or any alkaline drinks must be inhibited, and he should accustom himself to the daily use of the cold shower-bath, followed by strong friction over the whole body.

3. We can do little in this disease by mere medicines; but great attention should be paid to preserve a free state of the bowels, and from time to time the patient may use a mercurial, followed by a warm and tonic cathartic. In young patients, who approach to the anæmic state, the preparations of iron, cautiously exhibited, will be found useful.

But in the confirmed cases of persons advanced in life, especially where the patients have never exceeded in the use of stimulants, and above all, where the pulse is slow, with a tendency to faintness, or to attacks of the pseudo-apoplexy, our great reliance must be on the free use of wine or brandy,—for by such a proceeding alone can we hope to preserve or prolong the patient's life. Long fasting must be inhibited, and all fatigue or mental annoyance avoided. Such persons should always have some dif-

tuents of the system. Of the beneficial effects of pedestrian exercise, upon the weakened hearts of young men, I have seen the most remarkable examples in persons who had spent the summer walking through the Alps, when, during exercise they were under constant perspiration. Coupling this with the fact of the predominance of obesity in the colder countries, it seems probable that exercise taken in a warm climate is to be preferred in cases where we seek to prevent the accumulation of fat in internal organs. The researches of Schultze (De Adipis Genere Pathologico,) and of Schmidt (Jahrbuch, 1852, p. 147,) as quoted in the Medical Times, No. 113, may be consulted.

fusible stimulant at hand to which they may have recourse on any appearance of syncope, or of the cerebral sensations premonitory of an attack. They should be much in the open air, and if possible reside in a locality which combines the bracing effects of mountain and sea air, and as it is above all things necessary that their attention should be directed from the state of the heart, the routine and too often mistaken treatment of cardiac disease must be absolutely avoided.

It is truly singular how long life can be supported, and even the general health kept in an excellent state, by the adoption of these rules. In regulating the quantity of wine or brandy to be used, the physician must regard its effect rather than its amount. I have never seen injurious consequences to follow even a very free use of stimulants in this disease, and am in the habit of advising that they should be employed at every meal, or even during the intervals of meals. In the power of bearing stimulants these patients resemble those affected by the typhoid softening of the heart, some of whom, indeed, seem capable of using almost any quantity of stimulant without the slightest appearance of intoxication or determination of blood to the head.

That this disease frequently exists, and even advances to a considerable degree, although unrecognised by the attendants, or unperceived by the patient, is a fact of every-day occurrence. Under these circumstances, the following accident is too often seen:—An individual, previously supposed to be in good health, gets an attack,—it may be of gout, or of some local inflammatory disease. He is for a length of time debarred from his accustomed stimulus, and kept for days or weeks in the horizontal position. He recovers from the attack, whatever it may have been, and when all around are congratulating him on his restoration to health, he suddenly dies by a syncope, an apoplexy, an attack of cardiac asthma, or with a group of symptoms in which all these conditions may be said to be combined.

Cases of fatal metastasis of gout to the heart are probably much less frequent than is supposed. I myself have never met with such, but have known of many so designated, in which the nature of the attack was manifestly the sudden failure of the powers of the heart at the termination of a long fit of gout, in which the patient had been debarred the use of wine. In this way many a valuable life has been lost. It does not much matter whether these patients have the simply weakened, or the fatty and atrophied condition of the heart; for the indications of treatment, and the danger of the reducing system, are seen in either case.

It must never be forgotten that in the treatment of any patient suffering from local or constitutional disease, whose appearance or history renders the existence of a weakened heart probable, we must, in withdrawing the ordinary stimulants, proceed with extreme caution, and permit him to return to their use with the least possible delay. No good physician will withdraw stimulants without previously making a careful examination of the heart, even although nothing has awakened a suspicion of its being diseased.

Finally, we must bear in mind that there is danger from the patient being permitted suddenly to resume the erect position after convalescence. This is especially to be noted in persons advanced in life, and of a gouty habit.

In the preceding account of this disease, the occurrence of cerebral symptoms has been specified, as furnishing an important diagnostic of deficient power of the heart. It will be remembered that, in contrasting the cerebral attacks, under these circumstances, with those of ordinary apoplexy, the rarity of consequent paralysis was dwelt upon, yet such does occasionally occur. And the investigations of Dr. Law, on the connexion between cardiac and cerebral disease, must be here noticed.[1]

These researches have reference to the influence which a diminished supply of blood will have upon the brain in cases of cardiac disease; and though the anatomical changes which he has described are not necessarily produced in every case of weakened heart, or even in those instances of pseudo-apoplexy which are attended, as in Mr. Fleming's case, with recurrent and transient paralysis, yet we cannot but believe that the softening of the brain noticed by Dr. Law is, as it were, the last result of deficient arterial supply.

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[1] On Disease of the Brain dependent on Disease of the Heart. By Robert Law, M. D. Dublin Journal of Medical Science, First Series, vol. xvii. 1840.

The occurrence of white softening of the brain, as described by Rostan, was connected by him with disease of the cerebral arteries, and, doubtless, such a condition would have great influence in producing the lesion; but, as Dr. Law has shown that a similar affection may arise from disease of the heart itself, a probability is created that in some cases, such as those described by Rostan, a weakened state of the heart was really at the bottom of the malady.

The cases in which Dr. Law has observed this condition of the brain are as follows:—

1. Permanent patency of the aortic orifice, with regurgitation.
2. Contraction of the mitral orifice.
3. Permanent patency of the mitral orifice, allowing free regurgitation into the auricle.

It is plain that in all these examples the brain may suffer from deficient arterial supply, and although no case is given of softening of the brain, as a result of the fatty heart merely, we cannot doubt from the context, that Dr. Law would admit this condition also, resulting from anæmia of the brain, although there was no disease of the valves.

We cannot say why it is that cerebral anæmia in one case stops short at the production of syncope; in another, of syncopal coma, followed by transient paralysis; and in a third, goes on to the production of actual disorganization (the exsanguineous softening of Dr. Law;) yet no one can doubt that all these conditions, if not stages of the same morbid process, at least proceed from the one cause; and it is important to mention that, under these circumstances, a portion of the brain may be actually disorganized and broken down, and yet paralysis not be present. Thus, in a remarkable case of disease both of the aortic and mitral valves, in which the aortic opening was permanently patent, and the left ventricle much hypertrophied, Dr. Law found that the anterior lobe of the left hemisphere, where it rested on the orbital plate, presented a mass of softened structure, of about the size of the longitudinal section of a walnut. It occupied and destroyed the surface of the convolutions, but did not extend deeply into their substance. In this case there was no paralysis nor any weakness of either side.

The disease of the brain, to which Dr. Law has directed attention as arising from deficient arterial supply, may be met with in the following forms:—

1. Attacks of pseudo-apoplexy, or, as it might be termed, syncopal coma; sudden, transient, and not followed by paralysis.

2. Similar attacks, followed by paralysis, which is permanent, and may be attended with deficient mental power.[1]

3. Exsanguineous softening of the brain, unattended by paralysis.

4. Paralysis without manifest local disorganization.

I have introduced the observations of Dr. Law in this place, because they have so strong a bearing on practice in cases of fatty degeneration of the heart, where the question is as to the treatment of cerebral symptoms. To this point I have already alluded; but it has still to be settled as to whether in any case of apoplectic attacks which result from arterial anæmia, we would be justified in using the lancet. In the case communicated to Dr. Adams by Mr. Duggan, not less than twenty apoplectic attacks occurred during a period of seven years, and the treatment adopted was immediate venesection, and the exhibition of the strongest purgative medicine; and it is remarkable, that in the last and fatal attack the lancet was not employed, the patient having died before the attendance of his physician could be procured.[2] Yet this case is only valuable as showing that, in certain instances, the lancet may be employed with impunity. In determining this point of practice, it is probable that much will depend on the general condition of the system, the age of the patient, and the presence or absence of general anæmia. Where the heart's impulse is feeble, the pulse permanently slow, and when aortic murmur exists, we should avoid depletion. If we refer to the case by Dr. Fleming (see page 276,) it will be found that in one of the attacks, which occurred during Dr. Fleming's absence, the patient fell into the most extreme state of collapse, in consequence of the withdrawal of stimulants, and of the use of measures calculated to unload the head. Let us also consider the case which

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[1] A case of this kind is given by Dr. Law, Op. cit. p. 197.

[2] Dublin Hospital Reports, vol. iv.

I have given of weakened heart with aortic murmur, in which the patient was able to ward off the apoplectic attacks by hanging his head downwards, and we cannot help assenting to the principles indicated by Dr. Law, as to the treatment of cerebral affections arising from deficient arterial supply, whether they be connected with simple valvular lesion, such as occurred in Dr. Law's cases, or are attributable to fatty disease of the left ventricle.

An important case was communicated by Dr. Law to the Pathological Society of Dublin in 1845. The patient, aged 24, was attacked with hæmoptysis, dyspnœa, and cough, which led to the suspicion that he was suffering from phthisis. The physical signs, however, were only those of bronchitis. The heart showed signs of disease both of the aortic and mitral openings; for there was a double murmur at the base of the heart, and a single murmur to the left of the mamma. He left the hospital relieved, but was soon re-admitted in a state of stupor. The face was flushed, and the temporal artery throbbing; *but the action of the heart was diminished, and the abnormal sounds no longer audible.* The left side was partially paralyzed, and some convulsive attacks had occurred on the night before his admission. He was treated by the application of leeches and the use of calomel and James's powder, and in eight or ten days the paralysis had nearly disappeared, when, while at stool, he suddenly became comatose, and died almost immediately. The sinuses and superficial veins contained much fluid blood. The left corpus striatum was softened, and a quantity of grayish purulent matter covered the pons varolii, and was found in the left ventricle, and also extending to the spinal canal. Both the aortic and mitral valves were diseased, and the heart presented the rotund form which is common in mitral contraction. Another important case was communicated to the Society, during the same year, by Dr. Law, in which the mitral valves only were affected. The heart's action was extremely irregular, tumultuous, but weak; the countenance bluish, and the extremities cold. The treatment adopted consisted of stimulants, with nutritive diet, under which the patient improved until he happened to get an attack of pneumonia from cold. Dr. Law now reluctantly ordered venesection to the amount of eight ounces, *but when only half that quantity had been drawn,* *convulsions came on. On the following day the mouth was found drawn to one side; in fact the patient had become hemiplegic, and the heart's action was exceedingly feeble.* The stimulant treatment was now resumed, and with benefit, the hemiplegia disappearing. Subsequently another attack of paralysis occurred, after which the patient sank. The brain was not examined. The left ventricle of the heart was found dilated and hypertrophied; the auricle was dilated, as also the right ventricle. The mitral opening was narrowed by osseous depositions, and owing to the shortening of the chordæ tendineæ and thickening of the columnæ, it presented the appearance of being divided into two small openings.

It is probable that the use of cupping to the nape of the neck, with or without the scarificator, while, at the same time, we have recourse to stimulants, both internally and externally, will be proper. We should follow Dr. Fleming in the use of sinapisms over the region of the heart; and in cases where deglutition is impossible, we may use diffusible stimuli, such as brandy, ether, ammonia, or turpentine, by injection.

With a view to stimulating the muscular action of the heart, Dr. Law has employed the extract of nux vomica and the infusion of arnica. I have not used either of these remedies, and would merely remark that, with respect to the first of them, the dependence of the cerebral symptoms upon an anæmic state of the brain should make us less apprehensive in the adoption of the remedy. This will be quite consistent with the observations of Dr. Bardsley, which show that it is in the hyperæmic and irritative state of the brain that strychnine exercises an injurious influence.[1]

Among the local affections with which we find the fatty condition of the heart associated, various forms of bronchial disease are unquestionably the most common. We meet it in old persons who labour under bronchorrhœa, and who are liable to attacks of suffocative catarrh. It may be met with in very old and emaciated women who suffer from congestive pneumonia, or be associated with that form of atrophy of the lung and dilated bronchial tubes which follows on the cure of pleurisy, and the obliteration of the serous cavity. Lastly, it is found in ordinary cases of chronic bronchitis with Laennec's emphysema; and it is in this particular instance that we most often see injurious effects to result from overlooking the cardiac disease.

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[1] Hospital Facts and Observations. By James Lomax Bardsley, M. D. London: 1830.

Indeed, long before I had been aware of the frequency of the fatty degeneration of the heart, I had observed how very badly persons labouring under Laennec's emphysema bore a reducing treatment, even when labouring under inflammatory attacks. This peculiarity we were in the habit of attributing to the long deficient arterialization of the blood; but I am now convinced that in many of these cases the rapid sinking of the patient, under ordinary antiphlogistic treatment, is traceable to the existence of a weakened and fatty heart. Such a patient, from ordinary exciting causes, gets an attack of acute bronchitis, or, it may be, congestive pneumonia, and the practitioner, guided by the physical signs of pulmonary irritation alone, draws blood generally or locally, gives mercury and tartar emetic, inhibits stimulants, and at first is gratified by finding that the dyspnœa is relieved and the signs of pulmonary congestion or inflammation modified, or, it may be, removed. Soon, however, a large râle becomes diffused over the chest, and the patient, though cured of his pneumonia, gradually sinks into a state of asphyxia. In such cases the death is attributed to almost every cause but the right one.[1]

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[1] In many patients of this class there is a persistent crepitating râle occupying the postero-inferior part of one or both lungs, which is often nearly as fine as that in real pneumonia. The sound is clear on percussion; but this râle has been the cause of the loss of many patients; not that it is in itself of the slightest consequence, but from its being mistaken for the râle of acute pneumonia by practitioners who are ignorant of its nature, and who, on seeing the patient for the first time, take a condition which may have existed for months, or even years, for a recently developed disease.

CHAPTER VII.

ON THE CONDITION OF THE HEART IN TYPHUS FEVER.

It is not a little remarkable that, although the researches of pathological anatomists have been directed to the condition of the solid and hollow viscera in fever, and to the state of the blood and the elementary tissues, yet so little has been done with respect to muscular structure; and this observation will apply not merely to the state of the voluntary and involuntary muscles in fever, but also to their diseases, considered as local affections, independent of any essential malady. In the latter department, however, microscopic investigation, especially as applied to the conditions of atrophy and fatty alteration, has effected a great deal; but those alterations of muscular structure which are secondary to an essential state, and which, reasoning from analogy, should have a specific character in relation to the original disease, are still but little understood.

If we confine ourselves to the consideration of the muscular tissue in typhus fever, we find that the existence of an altered state of the voluntary muscles has been long admitted by authors. This condition is spoken of by Laennec as an adhesive softness, a gluey or fishy state of the muscles in connexion with the general phenomena of putrescence,—not of cadaveric decomposition, but of that dissolved state of the fluids which was held to be characteristic of the so-called putrid fevers. But on this subject opinion is by no means settled; and the question presents itself, as to whether this condition of the voluntary muscles represents the first stage of decomposition, or is a special secondary lesion, the result of the typhous condition.

Without at present discussing this question, it is to be remarked that even in fatal petechial fevers we rarely find the voluntary muscles affected, even in patients who exhibit the most important alterations of the viscera. In the fever of this country, at least, it may be safely said, that of all the organs of the body the system of voluntary muscles is that which exhibits the least apparent change on dissection.

If we now turn to the involuntary muscles, the conclusion is at once forced upon us, that between their state and that of the voluntary system there is no necessary correspondence or connexion; and it is certain that important alterations of the heart may be found, although the voluntary muscles appear to be perfectly intact.

A softened state of the heart, as attendant upon idiopathic fever, was noticed by Laennec, who observes, that he always thought it more marked in cases where the signs of alteration of the fluids were most evident. These signs, which were those formerly considered as marks of putridity, were, lividity and intumescence of the face, softening of the gums and internal membrane of the mouth, black coating on the tongue and gums, earthy aspect of the skin, distended abdomen, and fetid dejections; and although he does not express himself with distinctness on the point, he seems to hold that the condition of the heart is only to be taken as an example of that of the entire muscular system. This doctrine, which we have seen to be erroneous, was first corrected by Louis, who, in speaking of the softening of the heart in fever, observes, "that no similar lesion was found in any muscular organ; as all the muscles which preside over voluntary motion preserved amid the general disorder their natural colour and consistence."

The observations of Louis, so important, not only as establishing the existence and frequency of the lesion, but also as showing that it cannot be referred to any form of carditis, may now be studied:—

"The heart had less consistence than natural in twenty-four subjects. This diminution of consistence was slight in seven cases, and as when in this degree one might consider it less as a morbid state than as a variety of the natural consistence, or, as it is called, of its physiological state, I shall not consider these cases in what follows, and thus the number of patients we must examine is reduced to seventeen.

"The softening of the heart was, moreover, very slight in two of these cases. But as it was limited to the left side of the organ, we cannot consider it as the result of natural disposition; but there is a still more important reason for this opinion, viz., it happens sometimes, when the softening is considerable, that it is more so at the left than at the right. In the other cases it was universal and very marked, the heart was very flaccid, so that in many cases it had no precise form, but like a wet cloth retained any shape into which it might happen to be placed. Its substance had very little power of cohesion, was easily torn, and was very easily penetrated by the finger.

"At the same time that it was softened, the heart had less colour than usual in many cases; it was of an onion peel colour, which varied in intensity, and was generally livid and purplish on its surface as in its substance. The internal face of the ventricles and auricles was on the contrary, of a deep violet-red colour, which colour sometimes penetrated beyond the lining membrane, and appeared owing to an imbibition of blood, which it resembled more or less in colour.

"When thus softened and pale, the heart had no longer, when cut, the slightly moistened aspect it has generally, but it was, as it were, dry and unpolished, such as we have seen the liver to exhibit in analogous circumstances. Its size was not larger than usual, and it appeared smaller in two cases, (Obs. 14, 33,) and, therefore, it appears to me, we ought not to consider this as an effect of the softening of the organ, but rather as a natural *disposition* which existed in other patients likewise, in whom the heart presented nothing else remarkable, (Obs. 31, 39, 41.)

"Another fact, which it is important to notice, is this, that in nearly all the cases of softening, the walls of the ventricles were evidently much less thick than usual, those of the left especially, which were often only three lines thick. And as this diminution of thickness was limited to cases of softening, we must consider it as a morbid affection.

"If these facts are insufficient to enable us to discover the cause of the softening of the heart, at least they exclude the idea of one of those affections which usually induce a great number of diseases, viz., inflammation. For how can we allow that inflammation is the cause of an acute softening accompanied by a diminution of thickness, paleness of colour, a kind of dryness of the texture which is the seat of it? Such a supposition would truly imply a contradiction, and, as I remarked in relation to the softening of the liver, if we knew any cause of disease exactly the reverse of inflammation, it would be proper to refer this softening to it.

"Other considerations which I have already given in relation to the spleen support these affections. The walls of the heart, although more or less softened, showed no purulent secretion, nor was there any inflammation of the pericardium, which would have been the case had this softening been caused by inflammation. And in opposition to this opinion, we cannot produce cases of pericarditis observed after other acute diseases, inasmuch as softening of the heart was found in but two cases out of eight in which there was pericarditis.

"Moreover, the frequency and severity of the softening were much more marked according as the disease was more early fatal. Thus the heart was softened in nearly half of those patients who died between the eighth and twentieth days of disease; in a third of those who died during the following period; and in a somewhat smaller proportion among those who died afterwards. Besides, in seven cases in which the softening was extreme, not one was relative to individuals who died after the thirtieth day of the disease, and I found—

4 out of 17 patients of the first and second series.
3 out of 20 of the third series.

"Hence we see, that whatever was the degree of softening, the proportion of cases in which it took place in the different series of patients was very nearly the same; and it was, like that of the liver and spleen, more serious in those who died early in the disease than in those who died after the twentieth day, and we did not find it at its maximum in patients belonging to the fourth series. The rapidity of its development showed the extreme violence of the cause to which it was owing in certain cases, and, like other lesions of the same kind, it necessarily contributed much to produce death and hasten its arrival."[1]

Thus, it has been shown by Louis that in the continued fever, as met with in Paris, there is often a special alteration of the heart, differing in every respect from an inflammatory lesion,—an acute affection, attended with paleness of colour, absence of purulent infiltration, or of lymph on the pericardium. It was further found that the disease was best marked in proportion as the fever was more early fatal, being much more frequent in those who died from the eighth to the twentieth day than when the period was more protracted. If we reflect on these facts, and also on the general rarity of true local inflammation in fever, we must arrive at the conclusion, that the disease is not only not inflammation, but, to use the words of Louis, something which is the reverse of that state. It may be argued, that as there is a certain dissimilarity between the type of fever in Ireland and that on which the researches of Louis were based, the softening of the heart which is met with in these countries has nothing in common with that described by Louis; yet the advance of knowledge points to the conclusion, that however the continued fevers in the temperate regions of the earth may differ as to certain characters, yet that they are all closely related; that essentiality appears to be the rule, and its opposite the exception. Again, we may believe that their local alterations are secondary, that their origin is at least not inflammatory; and that they are inconstant in their seat and extent; to a great degree accidental; and always incompetent to explain the phenomena of the disease.

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[1] Louis, Recherches sur la Gastro-enterite. Bowditch's translation.

In most of the cases in which we observed the signs of softening of the heart, the patients exhibited a dark-coloured and abundant petechial eruption; the mouth was covered with sordes; the body exhaled the peculiar fœtor of typhus, while extreme prostration and stupor were generally present. In a large proportion of cases the secondary bronchial disease existed to a great degree, coming on in a singularly latent manner at about the fifth day of the affection, and although without much cough or prominent dyspnœa, often advancing so as to endanger life. The gastro-enteric symptoms, as they are termed, were generally less developed, though not unfrequently present. In many instances the disease occurred in young and robust men, and the bad symptoms were developed, at an unusually early period; the petechial eruption and the prostration appeared soon, and crisis was rare, yet the convalescence was generally satisfactory, and the restoration to health complete. The cases were, in other words, well-marked examples of the typhus of this country, and in a large number the disease could be traced to contagion. The health of these patients, previous to the attack, was in nearly every instance, good. There was no appearance of the scorbutic condition, and in the fatal cases it was almost always found that the voluntary muscles were red, showing the cadaveric rigidity well-marked, and in no respect diseased.

On these latter points it is important to dwell, as some have held that the condition of the heart observed in these cases was the result of putrefactive decomposition; but such a view cannot be admitted. In the cases observed by Louis, the alteration was confined to the left side of the heart; and our observations of the signs during life, and from dissection, establish that the left ventricle is the portion of the heart first and most prominently engaged. We have, it is true, observed the phenomena of softening of the entire heart, but all our results are confirmatory of those of Louis; and it would be a strange sort of putrefaction which would stop short at the left and not affect the right ventricle. But further, in many of our cases dissection was performed before putrefaction of the body had commenced; and as we have before remarked, the voluntary muscles were unaltered. Again, we often observed that the change of the muscular structure only penetrated to a certain depth, and was found to terminate by a well-defined line. Thus we could see the external portion of the ventricle affected to the depth, of from one eighth to the fifth of a line, and in such cases the musculi pectinati and columnæ carneæ remained unaffected.

But the great argument against this doctrine is the fact of our having been able to connect important physical phenomena during life with the softened condition as observed after death. We have traced the rise, progress, and retrocession of the weakened condition during life in cases which, from their close similarity with those that have presented this special alteration of the heart after death, furnish the strongest possible proof that the patients had laboured under, and afterwards recovered from, this peculiar alteration of the heart.

It may be noted also, that the signs of softening of the heart were by no means so commonly observed in the fever consequent on the famine of 1847, and this is the more remarkable from the fact that the scorbutic complication was then singularly frequent. My original observations were mostly made in the years 1837 and 1838, and it may be stated generally, that the phenomena of typhous softening of the heart appear to be under the same influences as those of other secondary affections, and that their frequency varies with the epidemic constitution.

Twelve years have now elapsed since the publication of my researches on the state of the heart in typhus fever, and since that period I have had no reason to change the opinions which were then formed, viz., that in certain cases of well-marked typhus fever we meet with a state of the heart which is not inflammatory, which is very different from that found in the voluntary muscles of such patients, and which is not to be attributed to cadaveric decomposition.

The condition now before us may be thus described:—The heart is little, if at all, altered in volume. It is generally of a livid hue, but this it may have in common with other internal organs, as is often seen in fever. It feels extremely soft, especially in its left portions, and the left ventricle frequently pits on pressure. Nothing remarkable is to be found as to the pericardium or endocardium, and the valves are unaffected. The principal change is found in the muscular structure, which is often infiltrated with an adhesive, as it were, gummy secretion. The left ventricle exhibits a singular appearance, for the traces of muscular fibre are lost, and the external layer, to the depth of the eighth of an inch, converted into a homogeneous structure, in which no fibre can be found. The colour of this altered portion is generally dark, and it resembles the cortical structure of the kidney. In some cases this change occurs in patches, varying in depth, and from a quarter to three-quarters of an inch in breadth. The change may affect the septum cordis and—but to a much less degree—the right ventricle. The internal network of fleshy bundles appears less engaged, and though these may be pale, their firmness seems but little altered. The right ventricle is almost always more firm and hard than the left, which may be so softened as to break down under a slight pressure. In one case both ventricles seemed almost equally engaged, and so great was the softening of the organ that when the heart was grasped by the great vessels, and held with its apex pointing upwards, it fell down over the hand, covering it like the cap of a large mushroom. Yet even in this case the left ventricle was more softened than the right.

In the remaining portions of the system we have not found anything which would distinguish the cases of typhus with softened heart from those in which the organ is unaffected. The most important of the secondary affections was the disease of the bronchial system, which was very frequent. As to the intestines, we sometimes found ulcerations of the ileum, while in other cases no such lesion existed.

Such is the condition of the heart in most of the cases in which the patient presented those physical signs which we attribute to the typhous disease. It will be seen that in many particulars there is a coincidence between our results and those of Louis. We have not, however, observed the thinning of the parietes of the heart, nor the dry and unpolished aspect of the cut surface. All the anatomical and vital phenomena of this affection point to the opinion that it is an example of one of the special secondary lesions of typhus, like the infiltration of the mucous glands of the intestine, and capable of retrocession, without consequent disorganization. We have observed no instance as yet of re-active disease occurring in the infiltrated structure, such as is seen in dothinenteritis, and, as we may believe, in the analogous affection of the lung. But we might suppose, when the function of the heart is considered, that before such a condition could arise, death would take place from debility of the organ; and it may be, also, that the muscular structure is less liable to re-active inflammation than that of the aggregate and solitary glands of the intestine. There are three important circumstances which must be here noted, as proving that in most cases the re-active disorganization does not occur. The first is the fact of the early restoration of the function of the organ in convalescence. Next, that we have occasionally observed, in cases of softening, an excitement of the heart immediately before death, and yet no marks of inflammation were discovered. And the third, that out of many hundreds of cases of weak and softened hearts observed during the last twelve years, we cannot adduce a single instance of organic disease of the heart which could be traced to any injury done pending the typhous affection.

If this softening of the heart be one of the secondary diseases of typhus, we should, as in the case of analogous lesions, observe something like periodicity in its phenomena,—at least we should observe that it was not coincident with fever, but rather intercurrent, arising and subsiding within some average period. Now we have found that in most instances the physical signs of weakness of the heart were developed at about the sixth day, and that after a period of eight days the heart seemed to be restored, at least so far as its contractile power was concerned, for in some cases slowness of action continued beyond this period. The average date of appearance of the signs was the sixth day, and that of cessation the fourteenth. This gives a period of eight days for the duration of the physical signs, but it is probable that in some instances the disease is developed before the sixth day. I have observed the commencement of weakening at least on the fifth day, and it may also be held that it occasionally subsides before the fourteenth day; for as the physical signs are our only means of detecting the rise and subsidence of the disease, it is neither likely that they would be well marked at its very first stage, nor indicate precisely the time of its disappearance. But even an irregularity in the period of its first development only makes the doctrine of this disease being a secondary affection of typhus the more probable.

In my original Memoir it was stated that we might divide cases of typhus fever, as met with in this country, into two great classes; in one of which progressive signs of weakening of the heart were to be observed, while in the other the action and sounds of the organ continued vigorous throughout the course of the disease. To this general division we still adhere, but it may be expected that in every epidemic, or in any extended period of observation, great variations will be found in the relative proportion of these two classes.

The researches which we have made subsequent to 1838 justify the following classification.

1. Cases exhibiting the signs of diminished power of the heart.
2. Cases in which the heart's action presents nothing remarkable throughout the course of the disease.
3. Cases of permanently excited action of the heart, yet without the physical signs or post-mortem appearance of inflammation.
4. Cases which are probably examples of simple debility of the organ, and which may be held to differ from those in the first class, in the absence of typhoid softening.
5. Cases which are generally of the short and relapsing fever, and rarely of maculated typhus, in which a bellows murmur is developed, which appears to proceed from a nervous or anæmic condition, rather than from an endocarditis.

Of these the first and the third may be taken as representing essentially opposite conditions, and the existence of either of them is not necessarily revealed by the state of the pulse, the warmth of the surface, or even the condition of general strength. For we may observe a hot skin and a pulse of average strength in cases where the action of the heart has progressively diminished, so as to become almost imperceptible; while, on the other hand, the heart may act with the greatest vigour in a patient who is pulseless, cold, livid, and covered with the darkest petechiæ and vibices.

The importance of this division cannot be overrated, as it involves questions of prognosis and of treatment of great weight; for the efficacy of stimulants in fever, and more especially the freedom with which they may be employed, seems remarkably to turn, if not on the actual state of the heart, at least on the existence of that group of symptoms of which its weakened and softened condition forms a part.

Since the publication of the researches of Louis, little had been done in this direction until the subject was taken up in the Meath Hospital; and our objects were, first, to determine the diagnosis of the softened heart in typhus; and next, to ascertain how far this knowledge could be made available in the treatment of the disease. It will be seen that the diagnostics which we shall presently describe are directly those of debility of the heart, and but inferentially those of softening; and the determination of a softened condition rests on our discovering the signs of debility occurring intercurrently in typhus fever. Yet the diagnostics between weakening with softening, and simple debility are not wholly indirect; because we find that between the signs of simple weakness and those of typhous softening there is this difference,—that the latter exhibit the phenomena of progression up to a maximum point, and then of retrocession, and that this double process occupies a period of many days, whereas in simple debility we observe not only that the heart seems to be at once weakened, but that its restoration may take place within the course of a single day. Again, the singular phenomenon of the progressive diminution of the rate of the heart during convalescence, when it may fall as low as 30 in the minute, again to rise gradually to its natural standard, is in all probability a condition peculiar to hearts that have suffered from the typhous infiltration. Doubtless, there is often difficulty, in the first instance at least, to say whether the patient has a simply weakened, or a weakened and softened heart; but the question is fortunately not one of great importance, at least so far as immediate action is concerned. And here we observe another illustration of the principle, that where a differential diagnosis is difficult or impossible, it is often unnecessary, so far as immediate practice is concerned.

Let us now review the physical indications of a weakened condition of the heart in fever. These signs point out two conditions: one of them is the progressive weakening, the other, the return of the normal condition of the heart. We may hold that the first series of phenomena indicates the progress of the typhoid infiltration, the second, its retrocession. In most cases the weakening of the heart is not preceded by any special sign. We seldom observe any preceding excitement of the organ, the action of which begins, as it were, to sink without notice. In a few instances I have observed a passing augmentation of impulse for a day or two before the establishment of the signs, but this may have been accidental; and the general rule is, that no increase of impulse, or other sign of excitement, except, in most cases, rapidity of action, precedes the depressed condition. We observe that the impulse becomes less and less distinct, the change being generally gradual, but in some cases more sudden. The loss of impulse is first perceived at the apex, and to the left side; and we frequently find that while it has ceased in this situation, it can be discovered under the ensiform cartilage. In the most extreme cases of loss of impulse, we cannot discover it even by careful examination, but this is rare; for in many instances in which the impulse appears to have completely subsided we may find it by examining when the patient is turned on his left side, or by pressing with the fingers in the intercostal spaces at the end of expiration, when we discover it like a feeble vermicular sensation. In most cases the diminution of impulse is attended with corresponding loss of sound; but it must not be forgotten that impulse and sound are not always proportionate or corresponding, either in the invasion or retrocession of the disease.

The re-establishment of the impulse generally takes place before that of the sounds, and in cases where all impulse has subsided, we observe its return first in the inferior sternal region, and next, under the left mamma. In most instances, however, the diminution and return of the first sound are accompanied with diminution and return of the impulse; but, in some the sounds may become distinct before the impulse returns; while in others the impulse re-appears long before the first sound has been fully restored. In one case we found that on the eighth day the sounds were not in proportion to the impulse: on the tenth, the impulse continued, but the first sound was totally absent; and on the eleventh day no impulse could be felt, and yet the first sound was distinctly audible. In another case, on the twelfth day, the impulse was less perceptible than on the day previous, but the first sound had more strength.

Many other illustrations of this might be given, and we must conclude that, in this state of weakness and softening of the heart, the force of the organ may be such as to give an impulse without sound, and again, that its contractions may cause a feeble sound although no impulse be perceived. Whether, in these cases, the impulse is occasionally similar to that which I have ventured to hint at in speaking of the fatty degeneration of the heart (see page 344,) is a question still to be determined. In both cases, at all events, the dynamic condition of the ventricle may be held to be the same.

It is hardly necessary to observe, that the value of want of impulse of the heart in typhus fever, as an indication of the change in question, depends entirely on the circumstance, that in the earlier periods of the case a distinct impulse had been observed. This will assure us that the want of impulse, under the circumstances in question, is not the natural condition.

Modifications of the Sounds.—We shall arrange these characters in the order of their frequency. The first, and unquestionably the most important, is the diminution of the systolic sound, which may go on to its complete extinction, or to such a degree as to give a singular predominance to the second sound. The progress of this lessening or extinction of the first sound follows the same law as the diminution of the impulse, that is to say, we observe it first as affecting the left side of the heart, and next, as spreading to the right; and the diminution or extinction of the systolic sound, as of the impulse, when observed at both sides of the heart, is indicative of an extreme degree of weakening.

Hence, as might be expected, we meet with the following combinations of circumstances:—

1. Feebleness of the systolic sound, causing predominance of the second sound, evident at the left side of the heart only: under the sternum no loss of proportion between the sounds can be discovered.

2. Cessation of the first sound over the left ventricle. We have then in this situation the heart acting with a single sound, and that sound the second, while beneath the sternum the double sound continues.

3. Cessation of the first sound at both sides of the heart, so that no matter where we examine, we find the heart acting with a single sound, which is the second.

4. In one or two extreme cases we observed the complete extinction of all sounds of the heart, and yet the pulse at the wrist could be perceived, and life was continued for more than thirty-six hours. One of these cases presented the greatest amount of softening of the heart that I have met with.

During the period of restoration of sounds the phenomena follow an inverse course, the first sound re-appearing under the sternum, soon to be followed by its restoration at the left side. Under these circumstances, too, we may occasionally observe that the progress of restoration of the systolic sound is from the base towards the apex. Thus, in a patient who presented well-marked signs of softening of the heart, we found that on the twelfth day, and when the pulse had fallen to 80, although the sounds were feeble at the base of the heart, they were quite proportionate; yet at the apex, and to the left of the ensiform cartilage, the first sound still predominated.

In the next series of cases the signs, though indicative of great weakness, are very different in character. Like the former, they are attended with diminution or loss of impulse, but the disease seems to act more on the entire heart, and we neither observe the predominance of morbid signs on the left, as compared with the right side, nor that of the second over the first sound. In these cases there is no extinction of either sound, but both are diminished in loudness, and become of a nearly similar character. To this condition I have given the name of the fœtal character, from the close resemblance of the phenomena to those of the heart of the fœtus in utero,—a resemblance which, especially when the pulse is acting at the rate of from 125 to 140, is almost complete. We have not yet been able to point out any anatomical difference between this condition and the more ordinary instance of diminution of the first sound, but it may be observed, that although we meet cases in which this character exists throughout, yet that in others both sets of phenomena occur at different periods, and, as it were, run one into the other. Coupling this with the fact that the two classes of cases are met with under the same epidemic constitution, and without any ascertained differences in the general symptoms, and, finally, that the treatment by stimulants is equally called for in either case, it may be concluded that there is a close relationship between the conditions in question.

Finally, we have observed in a very small number of instances a diminution of the second sound, while the first was but slightly, or not at all affected. It is not easy to explain this condition—possibly it is connected with a want of resiliency of the aorta itself; for in many cases the pulse is found to be remarkably compressible.

Such are the acoustic phenomena observed in these cases. Absence of valvular or aortic murmur is most remarkable, and everything points out a depressed, rather than an excited condition of the organ. Putting aside the negative proofs of this proposition,—the want of all physical signs of carditis,—there are two circumstances of great importance which strengthen this opinion. The rate of the heart's action, whether at the commencement or at the height of the disease, is variable, but in many cases it is very rapid, rising to 125, 130, or even beyond this. Now it is almost uniformly found that under the influence of powerful stimulation the rapidity of the heart day by day diminishes. This alone is a strong argument against the existence of any irritative disease of the organ, but it is not quite conclusive, for we should remember that in certain cases of asthenic pericarditis and endocarditis the best effects were found to result from the use of wine.—(See page 104.) But again, we observe the very curious phenomenon of the progressive retardation of the pulse during the convalescence of these patients. Thus we commonly find that at the period when we begin to exhibit stimulants the pulse may have been at 125 or 130, and the disease probably of seven or eight days' standing. After twenty-four or forty-eight hours' use of wine, in free doses, the frequency of the pulse is found to be lessened, and it descends from day to day until it reaches its natural standard, at which time, even if all symptoms of fever have not disappeared, we may safely hold the patient to be rapidly convalescing. Under these circumstances, however, the retardation of rate may go on, and the pulse fall, say from 80 to 76, then to 60, 50, and even so low as 30, yet all the time the convalescence of the patient is advancing, and his strength returning. Then the pulse begins to rise in its rate, and after a few days, as it were, finally settles at 72. During this time the action of the heart is perfectly regular.

It was suggested by Laennec that the rapidity of pulse occasionally observed in the convalescence of fevers depended on that softened state of the heart which he described as analogous to the gluey or fishy state of the voluntary muscles noticed in putrid fevers. On this point we have come to an opposite conclusion, for it appears certain that where the pulse is affected during convalescence in patients who presented the signs of softening pending the fever, slowness, rather than rapidity, is perceived.

With reference to the rate of pulse in convalescence, we may divide the cases into two classes. In one the pulse, after having attained its natural rate, remains unaltered. In the other, the sinking of its rate to a certain point, followed by its rising until the natural standard is attained, is to be observed. We find further, that in those cases in which during convalescence the pulse has continued rapid for an indefinite period, the fever has seldom been of the petechial or well-marked typhous character, and that in such cases the signs of softening of the heart had never been observed. There is a much greater degree of probability that quickness of pulse in convalescence, more especially if it occurs in a case where the pulse had come down after the fever, is to be considered as a sign of some existing local irritation. This may be the re-active inflammation of the intestinal deposits, such as probably occurred in the cases of imperfect convalescence noted by Dr. Cheyne.[1] I have more than once observed it to usher in the state of tuberculosis, the disposition to which had been excited by the preceding fever.

To fully understand the variations of these phenomena in fever, it will be necessary to compare a considerable number of cases; but before we commence this clinical examination it is proper to notice the observations of Professor Huss, with reference to an epidemic of fever which appeared in Stockholm[2] in December, 1841. The epidemic which he describes had many of the characters of the typhus of this country, but the predominance of cerebral symptoms appears to have been greater. The cases were divisible into three classes: first, those in which the cerebral symptoms predominated; next, those in which both cerebral and abdominal symptoms were present; and thirdly, a class in which the abdominal symptoms were the most prominent. The disease was not of a fatal character: out of sixty-four cases there were sixty-two recoveries. The general symptoms were those of typhus fever: the skin presented an eruption of maculæ, at first red, and afterwards becoming darker; and all the usual symptoms of prostration, typhoid delirium, non-critical epistaxis, stupor, bronchial congestion, sordes on the mouth and teeth; and in the abdominal cases meteorism and gurgling at the iliac fossa, and diarrhœa, with offensive stools, were observed. The usual sequelæ of fever occurred, and the general character of the disease was that of an essential affection in which the local diseases were secondary and variable. Professor Huss holds that the essence of the epidemic consisted in an alteration of the blood both in physical constitution and vital properties, the immediate result of miasmata. In this epidemic the phenomena of the heart were almost identical with those which have been now described. Thus in the cases of predominance of cerebral symptoms towards the close of the first period of the fever, which varied from five to nine days, the sounds of the heart, particularly the first, became enfeebled. As the disease advanced, the pulse would become small, thready, and unequal. Similar circumstances occurred in the abdominal cases, in the first stage of which, although the sounds of the heart were natural, they could be observed becoming feeble; in the second stage the pulse varied from 60 to 130; the sounds of the heart were at first feeble, then the first sound would become similar to the second, and gradually grow feebler until the second alone was audible. In such cases, as convalescence went on, the first sound was again heard faintly, then it became similar to the second, and the heart gradually recovered its normal rhythm with the inverse series of phenomena.

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[1] Dublin Hospital Reports, vol. i. p. 29.

[2] Observations sur la Fièvre Typhoïde qui a régne pendant les Mois de Décembre, 1841, et de Janvier, 1842, dans la Caserne du Corps de Gendarmerie de la Ville de Stockholm. Par Magnus Huss, Professeur de Clinique Médicale à l'Ecole de Médicine de Stockholm.—Gazette Médicale de Paris, Nos. xv. xx.

The close similarity between these phenomena and those which I first described will be still more apparent when we examine Professor Huss's observations on the use of stimulants in this epidemic. He gave muriatic acid while the pulse continued full and firm, and while the sounds of the heart remained normal, or while the first sound was only shortened. Phosphoric acid was indicated, when the pulse began to lose its fulness, and the first sound of the heart was so shortened as to resemble the second; and lastly, he used camphor and musk when the first sound of the heart became so feeble as to be heard with difficulty;

or, again, where the first sound had disappeared and the pulse was thready. Under these circumstances five grains of musk combined with a grain of camphor were administered every two hours, and as recovery took place the length of the interval was increased.

It would appear that in the great majority of the cases observed by Huss, there were, at some period of the disease, signs of softening of the heart. In the fever of this country, however, we may divide the cases into those with and those without any such condition, and the latter class might be subdivided into two groups, one of which presents an excited state of the heart, often more evident towards the fatal termination of the disease; while in the other the heart's action gives no evidence of either excitement or depression beyond the variations in rate which occur in every case of fever. In the cases of permanent excitement of the heart, however, there is no evidence of inflammatory action. We find neither the pericardial friction signs nor endocardial murmur; and dissection shows nothing but a heart which is not softened. It is difficult to declare the actual cause of this increased action, but it is probably to be placed in the same category as that of the various local but non-inflammatory excitements of organs in typhus fever. A familiar instance is the delirium with or without convulsions and coma, which may, as Louis has shown, arise without cerebritis; yet whatever be its nature, this at least is certain, that its occurrence is unfavourable, for there is hardly a worse prognostic in typhus fever than the co-existence of symptoms of general debility with an excited action of the heart, more especially if that excitement increases in the advanced periods of the disease. So that, considered with reference to a good prognosis, and having an eye merely to the state of the heart, we may arrange the cases as follows:—

1. The heart's action and sounds presenting nothing unusual throughout the course of the disease.

2. The occurrence of signs of progressive softening and recovery of the heart.

3. The existence of excitement of the heart, often increasing towards the close of the affection.

It is in this latter class that the use of stimulants is often found to fail; the nervous symptoms are frequently increased, and the rate of the heart augmented rather than diminished: and thus we may arrive at the apparently paradoxical conclusion, that the occurrence of a manifest local disease in an organ which is the centre of life, as in the case of the softening in typhus fever, justifies a more favourable prognosis.

Of this condition of the heart, the two following cases furnish good examples:—

CASE XXXV.—*Petechial Typhus Fever, with extreme Prostration; Continued vigorous action of the Heart; Failure of the Pulse after the eighth day; Death on the eighteenth day; Complete absence of organic lesion.*

In the spring of 1837 a woman, aged about 40, was admitted at an early period of fever. She presented the symptoms of an ordinary and rather mild case of maculated typhus. She had attended a person who died of peculiarly malignant fever, and also washed the clothes of the patient; and from the first had a presentiment of death which nothing could shake. She gradually became more collapsed; the surface was a violet hue; the countenance sunken; and the skin and breath cold. From the commencement of the second week, although the heart's impulse was strong, and the sounds remarkably distinct, no pulse could be perceived at the wrist. In this condition she remained for many days, during which time every possible stimulating treatment was resorted to. On the fifteenth day the surface was icy cold, but the heart continued to act with vigour; and as we could find no evidence of organic disease in any of the cavities, I determined to attempt the transfusion of blood. The operation was performed by Mr. Smyly; about six ounces of recently drawn blood were injected into the median basilic vein: a slight re-action followed, and the breath became warm. The pulse, however, did not return, and she died on the eighteenth day of the disease.

The most minute and careful dissection failed to discover any organic lesion in the brain, thorax, or abdomen. The arteries exhibited no obstruction; but the whole quantity of blood seemed much diminished, its consistence was pitchy, and its colour very dark. The wound in her arm was still gaping, and did not present the slightest appearance of adhesion or inflammation.

Of the nature of this typhoid excitement of the heart we are as yet wholly ignorant. We cannot say that the cause of failure of treatment in this case was, that the heart escaped the softening process; and it is only adduced to show that, in the most extreme form of adynamic typhus, we may find the heart acting with force all through the case, and even when the pulse is absent, and the breath cold.

CASE XXXVI.—*Maculated Typhus Fever, with violent action of the Heart; Death; No disease discoverable in the Heart.*

A girl, aged 16, who, it was presumed had been nine days ill, was admitted in November, 1839. She was delirious, and in a state of extreme prostration. The maculæ ran into large dark-coloured, almost black patches; and the whole surface was cold and livid. The tongue, lips, and teeth were covered with sordes; pulse 120, and so small and feeble that it was with the greatest difficulty it could be counted. *The heart acted with extreme vigour, and the sounds were loud and proportionate.*

On the following day the prostration was even greater: the face was livid, and the sordes of the mouth had become black; delirium and diarrhœa; pulse 126,—still weaker than on the previous day, while the action of the heart was, if possible, more vehement. She died on the thirteenth day, the pulse having risen to 146, and the action of the heart continuing to be violent and jerking up to the period of her dissolution.

Dissection fourteen hours after death.—A slight serous effusion was found in the pleuræ and in the pericardium, and the lungs were somewhat congested in their postero-inferior portions, but otherwise healthy. The heart was small, and in every respect perfectly free from disease. We were not permitted to examine the abdominal organs.

But a more common case of excited heart is one in which the general symptoms are those more usually seen in fever. We find that collapse does not appear until towards the close of the case; and that the patient presents the signs of local disease predominating in this or that cavity, or alternating from one to the other.

In such cases we may often find that the rate of the pulse is variable,—a circumstance long recognised as unfavourable; and again, that it becomes more and more rapid as the patient approaches the advanced periods of the disease. Imperfect crises are frequently observed, and the use of wine appears only to increase the rapidity of the heart, and in many instances excites or aggravates the cerebral symptoms. The impulse of the heart is jerking, and the sounds sharp and distinct; and these circumstances occurring at an advanced period are in the last degree unfavourable.

Let us now examine some cases which exhibit the more ordinary phenomena of softening of the heart in fever.

CASE XXXVII.—*Maculated Typhus; Absence of the first sound of the Heart; Extreme slowness of the Pulse during convalescence; Use of Wine in large quantities; Recovery.*

Matthew Hickey æt. 30, was admitted into hospital on the 15th July, having had fever for six days. Had been in the habit of drinking, but never to excess. He is the fifth of his family who have lately had severe maculated typhus; his countenance is much flushed; eyes suffused; maculæ abundant and of a bright red colour; tongue covered with a dirty brown fur, especially at the sides; great abdominal tenderness, particularly in the region of the liver. The chest, on percussion, yields a clear sound, and there are no stethoscopic indications of disease in either lung; the impulse of the heart is not perceptible; both sounds are audible, but the second distinctly preponderates; pulse 124; respiration easy and natural.

July 16. Slept well; bowels regular; tongue thickly coated and dry; convulsive respiration; skin pale and very clammy. Impulse of the heart is quite imperceptible, even when he lies on the left side; *to the right of the left nipple the second sound alone is audible;* pulse 120, rather feeble; on sitting up the impulse is not rendered more evident. Wine, 12 oz.; blister over the heart; beef-tea.

17th. There is still some abdominal tenderness; slept pretty well; respirations 28, interrupted by frequent sighing. He got yesterday 20 oz. of wine and a little brandy. The impulse is felt at the apex, but the sounds are by no means in proportion to its vigour; they resemble those of the fœtal heart; between the fifth and sixth ribs the sounds are barely audible. Wine 24 oz.; two glasses of brandy; blister to the scalp.

18th. Very restless; has not slept; frequently rises from his bed; passes urine freely; tongue red at the edges, and covered in the centre with a dark-brown fur; teeth coated with black sordes; lies on his back in a semi-comatose state; countenance collapsed and pale; maculæ very livid; respirations 32; on the back there are a few patches of ecchymosis; abdominal tenderness continues. The systolic sounds of the heart are very feeble, indeed, almost inaudible; the second is loud and clear, especially at a point central between the nipple and sternum; the impulse can only be felt by pressing on the intercostal spaces. Wine, 24 oz.; poultices to the abdomen.

19th. Skin cooler and moist; the respiration, although laboured, has lost its sighing character; the ecchymosed patches are fading; tongue cleaner; abdominal tenderness less; is more animated. Pulse 116; impulse of the heart the same as yesterday, the first sound is wholly absent, the second is distinct. Wine, 24 oz.; blister and poultice to the epigastrium.

20th. The countenance has lost the peculiar typhoid expression; the petechiæ are fading; breathing still laboured. Impulse of the heart quite imperceptible; the first sound is just audible; pulse 96. Wine, 18 oz.

21st. Passed a restless night; breathing much easier. He is quite sensible. Pulse 80, and of good character; the sounds at the upper portion of the chest are proportionate, but feeble; at the apex, and nearer the ensiform cartilage, the second sound still predominates. Wine, 12 oz.

23rd. Countenance more animated; skin cool; maculæ almost gone; complains of thirst; passes large quantities of pale-coloured urine. Pulse 76; impulse of the heart perceptible; sounds proportionate. Wine, 6 oz.

Discharged in a few days; pulse 60.

This case was one of extreme interest; the severity of the symptoms, the quantity of stimulants used, and the remarkable modifications of the heart's action, all combined to render the patient an object of the greatest attention.

The diminution of the first sound of the heart was the circumstance which led to the exhibition of stimulants boldly, at an early period of the case. We observed that on the seventh day the impulse was imperceptible, and the first sound was diminished. On the eighth, the first sound had disappeared, and although the other symptoms did not seem to call for active stimulation, we ordered wine in free doses from this indication alone, and the result justified the treatment. Here was a case of a young man of a good constitution, in which, from studying the action of the heart, we were able to anticipate the symptoms of general prostration, and by the early use of wine to prevent the fatal result which, it is almost certain, would otherwise have occurred.

The order of succession of the cardiac phenomena in this case was peculiar. We had—

1. The early subsidence of the first sound.
2. Both sounds audible, but with the fœtal character.
3. Predominance of the second sound.
4. Complete absence of the first sound.
5. Impulse imperceptible, with returning first sound.
6. The sounds at the base of the heart proportionate, while at the apex the second predominated.
7. The sounds natural.

The pulse, too, presented some interesting points for consideration. Within a period of twenty days its rate was as follows:

7th	day	of fever . .	124
8th	"	" . .	120
11th	"	" . .	116
12th	"	" . .	96
13th	"	" . .	80
15th	"	" . .	76
17th	"	" . .	60
18th	"	" . .	50
22nd	"	" . .	32
27th	"	" . .	56

In a few days it rose to 60.

In this case signs of the cardiac affection were observed at so early a period as the sixth day. It is probable that they had commenced even before this time. The early period of the fever, the youth and vigour of the patient, the heat of skin, suffusion of eyes, and tenderness of the epigastrium, all seemed to indicate that the time for stimulation had not arrived. But the coming danger was, as it were, foreshadowed by the signs of debility of the heart, and we were enabled to anticipate the adynamia by a timely use of stimulants.

In the following case, although many circumstances seemed to contra-indicate wine, we were led to its exhibition almost wholly from observing the phenomena of the heart.

CASE XXXVIII.—*Maculated Typhus; Signs of debility of the Heart predominating at the left side; Absence of Impulse; Free use of Wine; Recovery on the seventeenth day.*

A man, æt. 24, of strong muscular development, was admitted on the 25th of March, having been then nine days ill; his countenance was dull, stupid, and of a livid hue; eyes heavy and suffused; he was in a state of great stupor and prostration; decubitus on the back. Skin hot, dry, and covered with small livid petechiæ; tongue fissured, brown, and parched; had great thirst, and suffered much pain from pressure on the epigastrium; respirations 40, not laboured, and a few bronchitic râles could be heard in the left lung; the pulse 120, small and weak; the heart's impulse almost imperceptible, and the first sound so feeble as to be inaudible to the left of the mamma, but it could be distinguished between the mamma and sternum. Ten leeches were ordered to the epigastrium, artificial heat was applied to the extremities, and eight ounces of wine were administered.

26th. Slept well; countenance more livid; thirst insatiable; teeth covered with sordes; epigastrium less painful; extremities perfectly livid and cold. Pulse 116, small and weak; impulse of the heart quite imperceptible. The sounds are exceedingly feeble, and are almost inaudible below and to the left of the mamma, so that it is very difficult to distinguish between the first and second sounds, which, as it were, run one into the other; between the mamma and sternum they are stronger, and better defined; the second is much clearer than the first. If the rapidity of the heart was a little increased, the signs would closely resemble those of the fœtal circulation. Wine, 16 oz.

27th. Continued raving; involuntary evacuations; countenance improved; extremities cold and livid. Pulse 92, small, but distinctly stronger, and perfectly regular; the action of the heart can be seen between the fifth and sixth ribs, but can scarcely be felt; sounds of the left side remain as yesterday, those of the right are more distinct. Repeat the wine.

28th. Continued low muttering delirium; great prostration; involuntary passage of urine; extremities very cold, notwithstanding the use of artificial warmth; petechiæ livid; respirations 24; intelligence improved. Pulse 84; impulse less perceptible, but the first sound has more vigour. Wine, 16 oz.; musk, camphor, and ammonia mixture.

29th. Great improvement; extremities warm; petechiæ of a red colour; tongue cleaning; slept well; respirations 20. Pulse 84, firmer and steadier; when he lies on the left side the impulse of the heart is very perceptible, when on the back, it is less so, but more evident to the touch than on yesterday; sounds increased in strength and distinctness. Wine, 12 oz.; omit the mixture; to have beef-tea.

30th. Improvement continues, but the lower extremities become cold when the artificial heat is removed; the urgent thirst remains; tongue still brown. Pulse 72, fuller and stronger; pulsations of the arteria innominata are distinctly visible at the top of the sternum; impulse of the heart as before, but the sounds have improved in strength. Wine, 8 oz.

April 1. Convalescent. Pulse 72; full and compressible; heart's sounds distinct and natural. Wine, 4 oz.; beef-tea and chicken-broth.

Although this patient laboured under great prostration, and the petechiæ were livid, there were circumstances which we would, formerly at least, have held to contra-indicate the use of wine. He was young and of a robust constitution. His skin was hot and dry, and he suffered from thirst, a parched tongue, and tenderness of the epigastrium; but we relied on the cardiac signs, and the result showed that they did not mislead us. In this case we simultaneously leeched the epigastrium and commenced the exhibition of wine; the tenderness was diminished, but the thirst continued insatiable, and on the following day we did not hesitate to double the quantity of wine,—a practice to which we were led mainly by the increasing debility of the heart, and the development of the fœtal character of the sounds.

We have here a good example of the effects of wine in reducing the rate of the pulse, which on the tenth day of the fever and the third of the use of wine, fell from 116 to 92. At the same time the impulse, which had been wholly absent, became perceptible, and the sounds at the right side began to be restored. It may be stated, that where the systolic sound has been everywhere absent, its re-establishment will first be detected at the right side of the heart. The order of phenomena, in such cases, is as follows:—

1. Diminution of the systolic sound over the left ventricle.
2. Diminution of the same over the right ventricle.
3. Cessation over the left.
4. Cessation over the right.
5. Return of the systolic sound over the right.
6. Return of the sound over the left ventricle.

It is scarcely necessary to point out how strongly these facts corroborate the observation of Louis, that the typhoid softening affect the left in preference to the right side of the heart. But to this rule we have found a few exceptions, and have seen the right ventricle on dissection more softened than the left, which, however, was also decidedly engaged.

It may be laid down as a general rule, that there is no character of the pulse by which we can determine the presence or absence of the softened condition of the heart. The remarkable slowness which has been already noticed occurs after the impulse and sounds have been restored; and in many cases may not appear. We are unable to point out any difference in the preceding symptoms between the cases in which the pulse, during convalescence, falls far below the natural standard, and those where its retardation ceases on its arrival at the natural rate. In the best-marked cases of great diminution, or complete cessation of the first sound, the pulse, during the height of the disease, generally varies from 115 to 125; and it will often be found that when the rate passes 130 the phenomena consist in the cessation of impulse and the existence of the fœtal character of the heart's sounds,—a condition in which both appear to be affected, and one in which the sign of the heart acting with a single sound has not been observed. I have often thought that in cases of rapid action with feeble but equable sounds, the diagnosis of an actually softened state of the ventricle should be made with less certainty than in those where the pulse was below 125, with diminution or cessation of the first sound; for the existence of the fœtal character is often, as it were, temporary or fugacious, disappearing in the course of twenty-four or thirty-six hours, and not always followed by those progressive changes in the sounds of the heart which indicate a slower process of recovery. Yet, without seeking to establish that these cases are not examples of softening, we may hold that they indicate a state of debility, with, perhaps, less of the typhoid change. The existence, however, of this fœtal character should awaken the closest attention of the physician, for although the disease of the heart may have less of the element of organic change, and more of debility and functional derangement, yet there is something in its existence or production which cannot be referred to the mere rapidity of action. Both sounds are diminished, and the rhythm is unquestionably altered; and it will often be found that cases of this kind are of an irregular and anomalous character; that the stimulating treatment, though manifestly indicated, is not so certainly successful as in the ordinary examples of diminution or obliteration of the first sound; and that, as a general rule, our prognosis must be more guarded.

In the earlier periods of the disease, and before the heart has exhibited any distinct alteration, we are unable to say whether the subsequent condition will be that of the loss of the first sound or the production of the fœtal character. The two following cases illustrate this observation. But it is right to remark, that even in cases which present the fœtal character we may observe a certain disproportion between the sounds, the first, for a time at least, being less developed than the second.

CASE XXXIX.—*Maculated Typhus, with diminution of the first sound of the Heart; Use of Wine and Brandy.*

John Smyth was admitted into the Meath Hospital on the 19th of May, the tenth day of his fever. He is a strong, powerful man, who has been accustomed to drink ardent spirits, but has not been frequently intoxicated. At present he is very low: he was last night constantly getting out of bed; passes his urine under him; the petechial eruption is thickly diffused over his body; tongue dry, and red in the centre; intellect this morning clear; pulse 124, very small, and easily compressed; the impulse of the heart is feebly perceptible; the first sound very indistinct, the second clear; above the mamma the first sound is scarcely audible. Ordered, 8 oz. of wine.

May 20. Passed a good night, did not rave; respiration 36; retention of urine; extremities cold. Impulse of the heart imperceptible; second sound predominates over the first; pulse 124. Wine, 12 oz.; one glass of hot brandy punch.

21st. Slept well, no raving; petechial eruption livid; eyes suffused; respirations 36; tongue cleaner; retention of urine continues, requiring a frequent use of the catheter; bowels regular; the bronchial disease very severe. Pulse 112; the impulse of the heart is perceptible when he lies on his left side; the second sound predominates considerably. Wine, 12 oz.; dry cupping to the chest extensively; blisters to the region of the heart.

22nd. Had some sleep; no raving; countenance improved; eyes less suffused. Pulse 100, full and regular, *whereas before the additional quantity of wine given yesterday it was intermitting;* sounds of the heart feeble, the second still predominates. Wine, 20 oz. In consequence of his low state he was given 8 oz. of wine additional yesterday; beef-tea.

23rd. Passes his urine and fæces involuntarily; respirations 32; not so laboured; extremities warmed by artificial heat; great prostration. Pulse 84, small; the impulse of the heart is more distinct to-day; the first sound is still below par. Wine, 20 oz.; beef-tea; blisters over the heart and the nape of neck.

24th. Countenance much improved; slept well; when he is raised in bed he complains of giddiness; bronchial disease reduced considerably. Pulse 80; impulse of heart perceptible; the first sound is stronger. Wine, 20 oz.

25th. He had a quiet night; all the symptoms are improved. Pulse 80; very good strength; impulse of the heart natural, sounds proportionate. Wine, 16 oz.

26th. Scarcely any cough; sleeps well. Pulse 70, regular; phenomena of the heart as in the last report. Wine, 16 oz.

29th. Convalescent.

CASE XL.—*Maculated Fever, with severe gastro-catarrhal and nervous symptoms; Remarkable modification of the Heart's Action; Use of Wine.*

Thomas Cavanagh, æt. 15, was admitted on the 14th of April, being then three days ill: he had a few indistinct pale spots on the back; excessive thirst; diarrhœa, and tenderness of the epigastrium. There was slight cough, with abundant frothy mucous expectoration. Pulse 120, small, and easily compressed; but the impulse of the heart was strong, and the sounds distinctly heard over a large portion of the chest. The epigastrium was leeched, and effervescing draughts ordered.

April 16th. General symptoms continue; respirations 32. Pulse 126; impulse of the heart not so strong.

17th. Maculæ more distinct; the abdominal symptoms continue; respirations 36; some delirium. Pulse 120, weaker; impulse of the heart scarcely visible, but is quite perceptible to the touch; sounds are natural.

18th. Copious sweating after the use of a hip-bath; he is worse this morning; constant low delirium; countenance pale and depressed; less heat of skin; maculæ abundant, and becoming livid; tongue dry, brown; great thirst; considerable tenderness in the ileo-cæcal region. Pulse 132, still weaker; impulse of heart can be seen and felt; the sounds are exceedingly weak, *particularly the first, which is scarcely audible.*

19th. Debility increased; skin hot and dry; petechiæ universally abundant, and of a dark livid hue; respirations 30; less laboured; great thirst. The sounds of the heart exactly resemble those of the fœtus at the eighth month; an exceedingly indistinct impulse can be felt at the end of expiration. Wine, 3 oz.; arrow-root.

20th. Slept better, less raving; countenance improved; eyes less suffused; abdominal symptoms continue; respirations 28, interrupted by frequent sighing; sonorous and sibilous râles in the posterior portion of the chest; two small gangrenous spots on the left ear. Pulse 140, slightly improved in strength; impulse of the heart more perceptible, and its sounds can be heard to the right of the sternum. Wine, 3 oz.; arrow-root.

21st. Slept well, and is more collected; complains of extreme thirst; respirations 32; skin hot and dry; maculæ unusually abundant, and livid; one of the ecchymosed spots on the ear has vesicated; extremities warm. Pulse 132, fuller, more firm; impulse of heart as yesterday, but the sounds to the right of the sternum are not so distinct, particularly the first, which is remarkably feeble. Wine, 5 oz.

22nd. Symptoms as before; respirations 40. Pulse 125; no change in the heart. Wine, 5 oz.

23d. Raving continues; skin cooler; maculæ not so livid; cough worse, with much stuffing. Pulse 135. When he lies on the left side, the impulse of the heart is strong, first sound more distinct. Wine, 5 oz.

24th. The typhoid expression quite gone; eye clear and sprightly. Pulse 110, soft, and much improved; impulse and sounds still stronger. Wine, 5 oz.

Convalescent.

In a disease so variable in its complications as typhus fever, we should expect that, in its action on so mobile an organ as the heart, great varieties and modifications of phenomena may be met with. The study of typhus must convince the unprejudiced inquirer, that, exclusive of the well-marked secondary lesions which it is capable of producing, there are to be observed an infinite number of ephemeral functional, or of abortive organic changes, varying in their amount, seat, signs, and duration; and we cannot doubt that the heart, in common with other portions of the system, is subject to these influences just as much as the brain, the lungs, or the gastro-intestinal surface. These considerations help us to understand many circumstances connected with the action of the heart in fever, which are often obscure and anomalous, and which must not be lost sight of when we seek to obtain a comprehensive view of the subject.

As a general rule, diminution or loss of impulse is met with in connexion with the lessening or disappearance of the first sound. Yet to this rule some curious exceptions occur. It has been already observed, that in the return of the heart to the state of health, the restorations of the impulse and of the systolic sound do not always proceed *pari passû*, and we may observe anomalies and irregularities of this kind even in the earlier periods of the affection. In a very few cases we have noticed the absence of impulse as relating to the ventricular systole, but found that there was an impulse attendant on the second sound. The existence of a double impulse is by no means uncommon in many chronic cases, even without manifest lesion of the heart, but the want of the first or ventricular impulse, while that which attends the second sound remains, has, so far as I know, been only observed in the weakening and softening of the heart in typhus.

CASE XLI.—*Maculated Typhus Fever; Great prostration, with pulmonary and abdominal symptoms; Impulse of the Heart most distinct with the second sound.*

A girl, aged 13, having been already in fever for at least fifteen days, was admitted in a state of great prostration. Notwithstanding the advanced period of her illness, she was covered with an abundant crop of small dark maculæ. The face was livid and collapsed, and a copious stream of purulent secretion flowed from the nostrils. She had intense headach, continual delirium, and constant sobbing and moaning. The skin was hot; there was extreme thirst, and the epigastrium was tender; pulse 120, exceedingly feeble.

The first sound of the heart was scarcely audible, and attended with an impulse so feeble as to be barely perceptible. The second sound was very clear, and with it there was a distinct impulse. On the next day the first sound improved, and yet the character of impulse remained unchanged. During the following day, which was probably the seventeenth or eighteenth of the illness, the ventricular impulse began to appear, and the first sound was much improved; and on the twenty-first day the normal action of the heart was restored.

This was a very severe case. All the cavities appeared to be engaged; and the existence of livid maculæ at so late a period justified an unfavourable prognosis. The recovery, however, was perfect.[1]

The following case affords a good example of the occasional want of correspondence between the return of the impulse and of the first sound.

CASE XLII.—*Petechial Typhus; The sounds of the Heart feeble, but proportionate; Loss of Impulse continuing for five days with the progressive restoration of the sounds.*

A young woman was admitted on the eighth day of fever, which presented the usual symptoms of the epidemic. The petechial eruption had not yet appeared. The sounds of the heart were natural, and the impulse could be felt. It was observed, however, that the erect position brought on a tendency to syncope. On the twelfth day the sounds became feeble, though still proportionate, but the impulse had wholly disappeared; nor was it until the seventeenth day that it began to be perceptible, the pulse being then 76.

The slowness of the return of impulse, as observed in this case, is rarely seen; as in most instances the impulse becomes plainly perceptible for some days before the pulse has regained its natural standard.

In some cases the restoration of the first sound takes place equably over the region of the ventricle; in others, the returning first sound is only to be perceived at the apex, while at the base there is but the second sound, and a case has been noticed of restoration of the sounds from above downwards.

Again we find that, in certain cases, the alteration of the heart, whether it be its debility merely, or the special softening, does not always run the ordinary course of advancing regularly to a maximum state, and then as regularly subsiding. Thus the sounds may indicate progressive softening for two or three days, when a period of excitement takes place, which may last for twenty-four hours, and on its subsidence, the first sound may be found extinct, and the case go on in the ordinary manner.

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[1] The occurrence of a copious purulent discharge from the nostrils is a rare symptom in the typhus of Ireland. I have observed a few instances of it in which a sanious purulent fluid ran from the nostrils in a continued stream. The nose was swelled, and the appearance of the patient reminded the observer of a case of glanders.

In a few cases we observed a decided excitement of the heart following on its depressed condition, and continuing up to the period of death. Of this rare and untoward circumstance the following is a good instance:—

CASE XLIII.—*Petechial Typhus, with collapse and severe nervous and catarrhal symptoms; Great feebleness of the Impulse on the twelfth day; Vigorous action of the Heart for five days before Death.*

A woman, aged 45, was admitted on the eleventh day of fever. She was delirious, in a state of extreme collapse, with cold extremities, and a miserable pulse; decubitus on the back. The patient was constantly raving, and frequently uttered piercing screams. The face was flushed, and the expression wild and ferocious; the pupils were unaffected; the teeth and lips covered with sordes. She pointed to her head as the seat of distress. Thirst was urgent, and the surface of the trunk hot and dry, and thickly covered with livid petechiæ. The tongue was brown and fissured, and the pulse 136, small and weak.

The respirations were 46 in the minute, laboured and interrupted, and the vesicular murmur everywhere masked by intense sonoro-mucous rattles. The impulse of the heart was imperceptible, and the sounds could hardly be distinguished; but from the loudness of the bronchial rattle it was difficult to attribute the want of sounds to debility of the heart alone.

Wine was freely exhibited, and on the following day, although the coldness of the extremities still remained, the impulse could be perceived, and the sounds heard below the mamma: they were evidently much stronger than on the previous day. Pulse 140, slightly increased in strength. On the fourteenth day the pulse had fallen to 128, it was much fuller and stronger, and the heart could be felt pulsating over a surface of several square inches. The sounds were louder, especially the first. The wine, to the amount of an ounce every hour, was steadily continued, and it was found that after each dose there was a temporary, but manifest, improvement in the appearance of the patient.

From this time to the period of her death, which took place apparently from the increase of the pulmonary congestion or inflammation, the impulse of the heart continued vigorous, and the sounds distinct. Death took place on the eighteenth day.

The falling of the pulse on the fourteenth day from 140 to 128, after the free use of wine, was the only favourable circumstance which occurred in this case; for although this change after the use of stimulants was also accompanied by signs of returning vigour of the heart, yet these soon outstripped the limits which indicate a process of recovery, and a manifestly excited state of the heart succeeded to its depression. Such an occurrence, arising in the advanced periods of so severe a disease, led us to anticipate the worst result. And the observation of this case, and a few more of a similar kind, leads to the following rule:—*That the coming down of the pulse in fever, after the use of stimulants and the attendant progressive restoration of the force of the heart, is only favourable when the action of the heart stops short of a state of excitement.*

In this case the increased action continued up to the time when the patient died, but excitement following on the signs of commencing debility may be but temporary, as indicated by fulness and increased strength of the pulse, animation of the countenance, and augmented sounds. This state may continue for a period within twenty-four hours, when the sounds of the heart are more altered than they were before the temporary excitement set in; so much so, indeed, that in some cases the first sound is completely lost.

In most cases the signs of diminished power of the left ventricle are developed without there having existed any increased or irregular action which would draw attention to the state of the heart. In a very small number of instances a slightly increased, but ephemeral, excitement may be observed, yet this is rare, and the occurrence is probably accidental, for everything appears to show that the typhoid change of the heart, like that of other organs, is in its first stage an insidious and, as it were, silent process. In a few instances, however, we have found that before the signs of debility were established, the heart acted with increased force for a considerable period of time. These cases, however, were not examples of active hypertrophy, and we are not able to declare the cause of the increased force of the heart. The signs of carditis are wanting, and we find that, after the entire process of the fever has been accomplished, the heart, which has passed through the periods, first of excitement, or at least of continued vigorous action, and next of depression, with the diminution or loss of one of its sounds, re-assumes its natural action when the convalescence is completed.

The two following cases may be taken as illustrative of what has now been said, and they have an additional interest, as, in both of them we observed, during the existence of a stage of debility, that more rare occurrence of predominance of the first sound.

CASE XLIV.—*Petechial Fever, with Bronchitis and Diarrhœa; Vigorous action of the Heart up to the ninth day; Preponderance of the first sound on the sixteenth day; Use of Wine; Recovery.*

A strong muscular man, æt. 30, was admitted on the 11th May, being then nine days ill; he was abundantly maculated; well-marked bronchitic râles in both lungs; the action of the heart was vigorous, and both the sounds natural; pulse 108, full. The chest was cupped and blistered, and pills of blue pill and ipecacuanha exhibited.

May 13th. Severe diarrhœa. Pulse strong; both sounds are distinct and proportionate, but they seem as if distant. Omit the pills; poultices to the abdomen.

14th. The diarrhœa continues; maculæ abundant and florid. Sounds of the heart more feeble; the impulse is imperceptible, except at the end of expiration; pulse 100, strong. Saline mixture; arrow-root.

15th. The bronchial disease is more severe. Impulse quite imperceptible; both sounds feeble but distinct; pulse 100, feeble. Wine, 6 oz.; blister to the chest; pills of ipecacuanha, hyoscyamus, and carbonate of ammonia.

16th. Pupils contracted; bronchial disease continues; tongue glazed and red. Impulse imperceptible; sounds as before; pulse 92, a shade stronger than yesterday. Wine, 12 oz.; beef tea; antimonial mixture.

17th. Some diarrhœa; no vomiting; bronchial affection diminished; the petechiæ are not more livid; pupils less contracted; the tongue is becoming moist and pale at the edges; the wine was given warm. Impulse again perceptible; pulse 82; both sounds of the heart can be heard. Wine, 10 oz.; senega mixture; musk and camphor pills.

18th. Tongue improving; petechiæ fading. Sounds of the heart not so distinct as yesterday; *the second can scarcely be heard;* impulse perceptible. Repeat all.

19th. Slept well: diarrhœa continues. Sounds as yesterday; impulse imperceptible. Wine, 10 oz.; blister to the heart; bark mixture.

21st. General improvement; slept well; perspiration. *Both sounds can now be heard; they are feeble, but proportionate;* impulse imperceptible; pulse 72. Wine, 8 oz.

22d. Improvement continues. The sounds over the right cavities are proportionate; *over the left the first is much more feeble than the second;* no impulse. Repeat all.

23d. Skin cool; appetite good; no bronchial râles. First sound much more distinct; impulse plainly perceptible; pulse 72. Wine, 4 oz. Convalescent.

CASE XLV.—*Petechial Typhus, with Palpitation of the Heart and Bronchial Disease; Preponderance of the first sound of the Heart; Recovery.*

William Hawkins, æt. 34, was admitted into hospital on the 18th of October, being then eleven days ill. His illness commenced after a long exposure to cold, by rigors, succeeded by heat, and also by violent palpitations of the heart, which lasted for seven days. On admission the pulse was intermitting; there was a violent but intermitting action of the heart.

19th. (Twelfth day.) Maculæ abundant, severe headach, impulse of the heart feeble. *The first sound preponderates considerably;* abdomen tympanitic; the pulse regular, 100; he is constipated; a blister was applied to the abdomen; camphor, chalk mixture, and rhubarb wine, were ordered, and also a turpentine enema.

20th. (Thirteenth day.) The pulse was 104, stronger but intermitting; no impulse; the sounds more feeble, and the action intermitting with the pulse, *first preponderating considerably;* bronchitis in both lungs; was cupped, blistered, and ordered 5 grains of hydrargyrum c. creta every fourth hour. Was visited in the evening; the pulse and heart were regular. The bronchial disease became very severe; and for this he was repeatedly blistered, and the mercury pushed to slight salivation; the pulse and heart lost their intermission, *but the first sound preponderated all through.*

On the 24th (seventeenth day of illness,) he got 6 oz. of wine. On the twenty-first day the pulse was 64; no impulse of heart, the sounds became proportionate. On the twenty-ninth day the impulse was felt, sounds proportionate, pulse 64.

He left the hospital quite well.

CASE XLVI.—*Fever of a low character, following on an attack of gastric irritation; Absence of the second sound.*

A woman, aged 44, after having gone through a protracted bilious attack, was seized with the symptoms of typhus fever. She had great prostration, increased by a hypercatharsis, which followed on the treatment so commonly adopted by the lower orders in this country, of using a strong dose of a saline cathartic at the commencement of fever. Her general symptoms were those of petechial fever of a bad character with severe nervous symptoms, diarrhœa, bronchial irritation, and large livid petechiæ. On the eighth day the pulse was 114, and weak, and the heart acted with a single sound. On the ninth day we ascertained that this sound was the first. The pulse was then 170, and extremely weak. The systolic impulse existed, though much lessened in force, and the first sound which, though diminished, could be heard at the apex, and about one and a half inches upwards, suddenly ceased at the base of the heart. Next day the first sound was very evident, but there was no second sound. She sank on the eleventh day. We failed in obtaining a dissection.

It has been already suggested that there is some connexion between the force of the arterial pulsations and the character of the second sound. In most cases, indeed, where the second sound is feeble, we find a remarkably weak pulse; and a case will be given, in which with a strong and throbbing pulse, the second sound was singularly loud and distinct, although the first had become obliterated.

Upon the nature of the excitement of the heart, produced as an intercurrent condition in typhus fever, we as yet have but a negative knowledge. It is not a carditis, and, as has been already remarked, we can only hold it to be analogous to other instances of functional lesion so common in the course of the disease. I incline to the opinion that it is more liable to occur in the female subject. The instances in which we have met with it may be arranged as follows:

1. Permanent excitement of the heart, attended with general collapse, coldness of the surface, and failure of the pulse. See Case XXXV.

2. Ephemeral excitement arising in a case which has already shown the signs of commencing debility of the heart, but soon being succeeded by well-marked signs of weakness and softening. See Case XXXVII.

3. Increased and excited action, arising in the advanced stages of a case in which the heart has gone through the processes of weakening, softening, and restoration. See Case XLIII.

The occurrence of any of these forms should always lead to an unfavourable prognosis. The second of these cases is the least formidable, but there is nothing which should arouse our apprehension more than the occurrence of an excited heart in the advanced stages of fever, especially when it is attended with the general symptoms of debility, and with rapidity, feebleness, or failure of the pulse.

Strongly contrasted with the condition of excitement is that in which the impulse and systolic sound are lost. It cannot be too much insisted on, that this condition, though marking an extreme degree of weakness of one or both ventricles, is not necessarily indicated by the strength of the pulse, which may preserve a fair degree of strength and volume, even where the softening of the heart goes on to the extinction of the first sound. Hence the physician, in determining the state of the circulation in fever, must not be content with the examination of the pulse alone, but combine with this a careful investigation, repeated from day to day, into the force of the impulse, and the character of the sounds of the heart. The following may be taken as a fair specimen case of failure, and ultimate restoration, of the sounds and impulse.

CASE XLVII.—*Severe Maculated Fever; Delirium; Fœtal character of the sounds of the Heart; Use of Wine in large quantities; Recovery.*

Patrick Quin, æt. 20, was admitted on the 27th of February. It was stated that he had been ill but five days, but his appearance was that of a person after a much longer period of fever; he was collapsed, cold, stupid, and covered with an abundant crop of livid maculæ; prostration extreme; eyes diffused; tongue covered with brown sordes; pulse 125, small and weak; heart's action feeble; respiration hurried. He was ordered 4 oz. of wine.

February 28. Violent delirium during the night; he is now in a state of collapse, lying on his back; constant jactitation; subsultus; cold extremities; retention of urine. Pulse 132, soft, small, and variable; heart's impulse imperceptible; sounds weak but defined; the pulsations in the carotids very feeble. Wine, 24 oz.; blister to the head.

March 1. Slept well; in other respects is nearly the same, but is more easily roused; less suffusion of the eyes; considerable subsultus; he passed urine involuntarily. Pulse 120, a shade stronger than yesterday; *the sounds of the heart are similar to those of the fœtal circulation.* Wine, 24 oz.; turpentine enema.

2nd. Slept well; tongue moist; respirations 30; maculæ fading; extremities warm. Pulse 130, fuller and stronger; heart's action stronger, and its sounds much louder—they approach to their natural character. Wine, 14 oz.

3rd. Violent delirium throughout the night; skin hot; pulse 104; heart's impulse stronger. Wine, 12 oz.

4th. No change; great thirst. Pulse 106. Wine, 16 oz.

5th. Patient worse; countenance more collapsed; violent delirium; picking of the bed-clothes; subsultus; sighing; contraction of the pupils; incontinence of urine; skin hot and dry; mouth covered with black sordes. Impulse of the heart plainly perceptible; second sound much louder than the first; pulse 120. Wine, 16 oz.; turpentine draught, with camphor, musk, and opium mixture; beef tea; swathing with flannel.

6th. Generally improved; slept well; much more sensible. Pulse 106. Wine, 16 oz.

7th. Complains of great thirst; extremities warm; maculæ bright red, and less abundant; pupils natural; tongue moist. First sound of heart much stronger; pulse 96. Wine, 16 oz.

8th. Great improvement; desire for food; skin cool. Heart's action nearly natural; second sound much improved; the abdominal aorta can be felt throbbing with force. Wine, 16 oz.; omit the medicine.

9th. Skin cool; slept well. Impulse of heart vigorous; strong action in the arteries of the neck and in the abdominal aorta; pulse 88, strong and full. Omit the wine.

12th. Convalescence perfect. Sounds and impulse of the heart natural; pulse 72.

The foregoing cases will give a sufficiently accurate idea of the signs furnished by the ordinary examples of typhoid softening of the heart, and also of the general character of typhus fever, as met with in this country. In a large proportion of our cases the fever could be traced to contagion; and termination by crisis, especially where the signs of softening of the left ventricle were developed, was rarely if ever observed. In most instances the signs indicated that the disease predominated in the left ventricle, so that a not uncommon condition was, that the second sound was all that could be heard on the left side, while under the sternum both sounds were feebly audible, although the second was predominant. In more decided cases, as might be expected, the systolic sound was everywhere absent, and in a few instances the singular condition was observed of the continuation of the pulse at the wrist for thirty-six or forty-eight hours after all sounds and every trace of impulse had disappeared from the heart. Such cases, as might be expected, proved fatal. Are we to attribute this result to the lesion of the heart alone, or rather, consider the cardiac affection as consequent on the extreme malignity of the disease?

Hitherto we have considered the typhoid softening of the heart more as a guide to prognosis and treatment than in itself a source of danger; yet it is difficult not to attribute danger to a condition of the heart in which both ventricles lose so much of their contractile power, and this especially when we recollect the frequency of complication with the secondary disease of the lungs. The character of respiration termed cerebral has frequently been met with in these cases; yet there appear good reasons for thinking that it is analogous to that which occurs as a symptom in chronic debility of the heart. This we have noticed in the chapter on Fatty Degeneration. And it is certain that the "cerebral respiration" in typhus has not been found to contra-indicate the use of wine. May we not, then, believe, especially when the researches of Drs. Hudson and Law are borne in mind, that the brain in typhus may suffer from the weakened condition of the left, and the lungs from that of the right ventricle.

CASE XLVIII.—*Maculated Typhus; Loss of Impulse on the twelfth, and of both sounds on the thirteenth day; Death by syncope on the fifteenth day; Extreme softening of the Heart.*

A strong and healthy-looking man, aged 28, was admitted on the sixth day of fever. He had been living in a house with three persons labouring under maculated typhus, when, after exposure to cold, he was attacked with the first symptoms of the disease. On the day following (the seventh) he was much prostrated. Skin very hot and dry, and beginning to exhibit large maculæ. Pulse 98, full and strong; respirations 36; and over both lungs a fine muco-crepitating râle could be heard. The impulse of the heart was jerking, and both the sounds were distinct but feeble. No important change occurred until the ninth day, when the jerking impulse ceased. On the eleventh the impulse was weak, and although both sounds could be heard, they were very feeble, and the second predominated. All the bad symptoms now increased. The maculæ were more abundant. Pulse 116, thready and compressible, while the posterior portions of the lungs were much congested. Over the left cavities of the heart both sounds were lost, but they could be found at the right side. The impulse had ceased. Next day all sounds of the heart had disappeared. Pulse 144. Skin hot, and the maculæ very abundant. On the next day the pulse had fallen to 128, and was stronger, and a shade of the sounds of the heart might be discovered at the right side. This favourable change appeared to go on. The pulse came down to 112, and the general appearance of the patient was greatly improved. He took food with relish; when at midnight he had a sudden change for the worse, and died in a few hours, apparently in a state of syncope.

Dissection, twelve hours after Death.—The voluntary muscles, with the exception of the pectorals, were perfectly healthy, but the latter was soft and gluey. Serous effusion was found in the right pleura, and to the amount of six ounces in the pericardium, which was otherwise healthy. Both lungs were much congested in their posterior portions. The heart was pale, and its muscles perfectly relaxed. When laid on the table it spread out flatly, and when held up by the great vessels it fell over the hand like a cap. The substance of both ventricles broke down into a pulp on the slightest pressure. The heart felt like a soft anasarcous limb, and the surface, as exposed by an incision, distilled everywhere an adhesive matter. It was hardly possible, even with a magnifier, to detect the traces of muscular fibres in the ventricular walls. The large vessels and the abdominal viscera were healthy.

In this case, as in that next to be described, the right ventricle was found to be even more softened than the left. We may conclude, both from dissection and the study of the physical signs, that this circumstance is one of very great rarity.

CASE XLIX.—*Adynamic Typhus, with severe nervous and pulmonary symptoms; great debility of the Heart, with preponderance of the first sound. Death on the twelfth day.*

A woman, aged 40, presented on the eighth day of her illness the symptoms of extreme prostration and anxiety; the surface was covered with large dark maculæ, many of which were elevated; respiration 40; pulse 140, and full. The patient had been bled before admission. On the ninth day of her illness she had subsultus tendinum and low muttering delirium, with congestive râles over the chest. The impulse could scarcely be felt. At the apex both sounds could be heard, but the first greatly preponderated, while at the base the second sound was inaudible. On the eleventh day she was found lying on her back, in a semi-comatose state; the maculæ had run into large livid patches; the pulmonary congestion had increased; and every attempt to swallow brought on a suffocative attack; the pulse was 160, and extremely weak, and the slightest attempt to sit up was followed by the most alarming faintishness. The impulse was imperceptible; the first sound still preponderated. She sank on the following day, the twelfth, without any struggle, no treatment having had the slightest beneficial effect.

Dissection.—The heart alone was examined. About 6 ounces of pale-coloured serum were found in the pericardium; the heart was pale, flabby, and so exceedingly soft that it broke down under the slightest pressure; and we observed that the right ventricle was more softened than the left.

CASE L.—*Petechial Fever: Prostration; Varying state of the Heart and Pulse: Death.*

A woman, aged 30, of good constitution, was admitted on the sixth day. The petechiæ were abundant and very livid. She had diarrhœa, delirium, and want of sleep. On the ninth day, after the use of wine, the pulse rose from 120 to 130; and on the twelfth day it was found necessary to omit the stimulant from its producing great nervous excitement. The worst prognosis was made. The prominent symptoms were delirium, a diphtheritic exudation in the fauces, the sighing, so-called cerebral respiration, and extreme debility.

During the whole case the condition of the heart was singularly variable. On the eighth day the sounds were more distinct than on the seventh, and the pulse 120; next day the pulse was 130; the impulse scarcely perceptible, and the sounds, though proportionate, had again become feeble; then for one day the action was so irregular that it could not be analyzed, and all impulse ceased, but on the following day the pulse was 124 and full, yet the impulse was wanting, and the sounds extremely feeble. The impulse returned on the thirteenth day; the sounds were stronger, and the pulse came down to 100. In two days it rose to 120, then to 130; and on the day before death the state of extreme irregularity, as on the eighth day, returned. The first sound of the heart was then scarcely audible, while the second was distinct. She died on the nineteenth day.

Dissection, twenty hours after death.—The general muscular structure was healthy, and the lungs showed some congestion in their posterior portions. The pericardium contained about half a pint of straw-coloured serum, but was otherwise healthy. The heart was small, and showed softening of the left ventricle, though not to the same amount which we observed in other instances. The muscular fibres could still be seen on the cut surface, yet the structure had a more homogeneous appearance than natural, and exuded a glairy, semi-gelatinous fluid. The intestines were free from ulceration; but the mucous membrane of the jejunum and ileum was much congested and softened.

CASE LI.—*Severe Maculated Typhus, complicated with intense pulmonary irritation, the sounds of the Heart having the Fœtal Character; Employment of Wine; Death.*

John Harris, of full plethoric habit, had always enjoyed good health, and although in the habit of taking whisky, never drank to excess; has had fever for six days; his chest and arms are covered with well-defined bright-red petechiæ; complains of much pain in the head and dimness of sight; tongue furred, epigastrium very tender on pressure, bowels constipated; passes small quantity of urine; pulse 96, and full; respirations 28; some wheezing and sibilant ronchi are heard anteriorly; face very much flushed. He was ordered effervescing draughts.

In four days the pulse rose to 116; the sounds of the heart were very feeble, and on the tenth day closely resembled those of the fœtus in utero.

From this period the patient got worse. The bronchial disease became intense and general, so much so, that it was hardly possible to make any accurate observation of the sounds of the heart; the impulse, however, was imperceptible; the pulse became intermitting, and increased in frequency to 120, and on the next day to 136. On the sixteenth day his extremities were cold. He died on the following day, the seventeenth of his disease. In this case active stimulation was employed. The patient got nearly eighty ounces of wine; he was dry-cupped and blistered, and used emetics, from which he experienced great relief some days before his death.

Post-mortem Examination, eleven hours after death.—The heart was of its natural size, livid, and extremely soft, pitting on pressure, particularly over the left ventricle; some white patches could be seen on the right ventricle; the lining membrane of the left auricle presented nothing remarkable. The left ventricle was divided from its base to its apex; the muscular substance presented a very singular appearance, not a trace of fibre being visible; and for more than two thirds of its length a layer, of one-eighth of an inch in thickness, was found, presenting a darker colour, and of more homogeneous appearance. The substance of the ventricle was infiltrated with a gummy matter, causing the fingers to stick together, and the structure had some resemblance to the cortical substance of the kidneys; a transverse section gave the same appearance. The network of fleshy fibres exhibited more firmness, though similar in condition. The posterior columns seemed but little altered, being only pale, their firmness remaining perfect; the same might be said with respect to the anterior. The right ventricle was harder and firmer, and did not exhibit the same aspect as the left; the right auricle contained a coagulum; nothing remarkable was observed as to the colour of the lining membrane; the septum cordis was softened and livid. On examining the abdomen no disease presented itself. The ileum was perfectly healthy; no enlargement of the glands.

CASE LII.—*Maculated Typhus, with severe nervous symptoms; Predominance of the second sound on the sixth day; Complete absence of the first sound on the tenth day; Death; Softened state of the Heart; Ulceration of the Ileum.*

Richard Cashel, æt. 46, admitted 5th November; six days ill; he complains of pain in the back, neck, and extremities; considerable prostration; maculæ abundant, of light colour on the chest and abdomen, but much darker on the back; slept very little last night; raved a good deal, but was not violent; has no headach; pupils slightly contracted; very little cough, unaccompanied by expectoration; stools thin and watery; abdomen soft and tender on pressure; great thirst; tongue brown and dry in centre; teeth covered with sordes; pulse 116, rather feeble; respirations 28; auscultation detects slight bronchitis in both lungs. When the patient lies on his back the impulse of the heart cannot be felt, but it becomes perceptible when he turns on his left side; both sounds are audible, and the second predominates slightly over the first; ordered an anodyne enema and 8 ounces of wine.

November 7th. Raved much; skin hot and dry; maculæ dark; respirations 28. Pulse 116, as yesterday; the impulse of the heart cannot be felt to-day; sounds more feeble than yesterday, scarcely audible above the mamma and to the left; more so at the sternum; the second preponderates. Repeat the wine.

8th. No raving; extreme prostration; considerable fœtor from the body; maculæ very dark-coloured; sordes of the mouth abundant; is unable to raise himself without assistance. Pulse exceedingly feeble and irregular, being from about 116 to 124; no impulse of the heart; the systolic sounds very feeble, and almost inaudible to the right of the nipple; the second is still loudest. Wine, 16 ounces; beef tea.

9th. Was very restless all night, picking at the bed-clothes, and muttering constantly; passes his water under him; lies on his side; maculæ very abundant, and quite livid on the back. Pulse 120, exceedingly feeble, and disappearing on the slightest pressure. When he lay on his left side the impulse could not be felt, but when he turned on his back it was found to be vigorous; *the double sound of the heart was completely lost, the distinct clear " rap " of* *the second alone being heard; most distinct also at the base of the heart.* Wine, 20 ounces; brandy, 2 ounces.

10th. Lies on his back; mouth wide open; constant spasms of the muscles of the lower jaw; constant moaning; is in profuse perspiration; excessive fœtor from the body; respiration 40; stools involuntary. Pulse 150, exceedingly weak and irregular; impulse of the heart evident; in consequence of his moaning no accurate observations of the sounds could be made.

Died at one o'clock, P.M.

Dissection, twenty hours after Death.—The body was more than usually livid; the petechiæ were pale on the fore part of the body, but very dark and livid on the back; abdomen tympanitic; the pericardium contained about half a pint of straw-coloured serum; the heart was of large size, and so extremely flabby that it was capable of retaining any shape in which we placed it; the right cavities were softer than natural, admitting the fingers through their walls without much resistance; in the muscular structure of the left cavities, however, this change was much more remarkable; they were so exceedingly softened that the weight of the finger was almost sufficient to penetrate their walls; they were easily torn, and the edges thus separated had no longer the moistened appearance, but seemed as if quite dry. The septum cordis was equally softened; there was some dark fluid blood in the right cavities. The stomach presented some red patches, slightly elevated; towards the pylorus the mucous membrane was thickened and softened, and easily removed by the handle of the scalpel. The duodenum was tolerably healthy, having only in two or three places slight blushes of inflammation. The ileum was more extensively involved; this was particularly observable in the last two feet of its length. Near to the ileo-cæcal valve there were five circular ulcerations; the superficies of the ulcers were covered by a delicate membrane, beneath which there was a yellow-coloured fluid, resembling pus; the largest was about the size of a silver penny: round these infiltrated points the intestine was much inflamed, and several minutely injected capillaries were seen ramifying around them, but these could not, even by the aid of a good lens, be traced into the ulcers. When the membrane was removed under water, and the puriform matter washed off, a decided depression was left, at the bottom of which was easily seen the muscular coat of the intestine: dispersed further throughout the intestine were several of the elliptical patches. The glandulæ aggregatæ were prominent in many places.

One of the most remarkable circumstances in this case is the varying condition of the impulse, as seen in the reports of the few days preceding dissolution. Let us also note that on the day before death there was a good impulse when the patient lay on his back, and yet the first sound of the heart was completely lost. In these cases we commonly observe the phenomena of impulse without corresponding sound, and of sound without impulse. In the state of the pulse and the physical signs in this case,—and even more especially in that which precedes it,—we may see something like a succession of ineffectual struggles of the heart with the disease which has attacked it. A pulse varying in frequency, within short spaces of time, has long been considered as an ill omen in fever; and in Case L. we observe this condition well-marked on the seventh, eighth, and ninth day of this fatal case. The phenomena of the heart were simply those observed in many instances in which there was a favourable result, and under the use of wine the pulse, after having been one day irregular, fell frequently from 130 to 124, 112, and 100; and at this time the impulse re-appeared, and the sounds became stronger. But in two days it again rose to 130, then fell to 120, with excitement of the heart and proportionate sounds; and the next day the first sound was scarcely audible, while the second was distinct and clear. Death occurred on the following day, and the left ventricle was found to a certain degree softened.

Are we to explain such phenomena by assuming that a new attack of the secondary typhoid disease takes place? or has there been the commencement, at least, of re-active inflammation in the substance of the heart itself? Future researches must determine these points, and how far the varying innervation of the heart, without organic change, may influence the phenomena.

CASE LIII.—*Adynamic Typhus; Vibices; Delirium and Hæmoptysis; Death on the twenty-seventh day; Softening of the left ventricle.*

A young man was brought to hospital in November, 1839, having been already three weeks in fever. He was in a state of extreme prostration. The surface was cold, and covered with large vibices. The mouth was full of sordes. He had cough. Pulse 130, very weak. The impulse of the heart could scarcely be felt, and the second sound was predominating. On the day following his admission the surface had become warm, but the impulse could only be found when he turned on the left side. The sounds over the right cavities were distinct, and yet the second predominated. On the twenty-third day he expectorated a large quantity of dark-coloured blood, followed by a copious discharge of muco-purulent and bloody secretion from the chest. He soon fell into a semi-comatose state. The signs of intense pulmonary congestion increased; the pulse ranged at about 124; and after a passing state of high nervous excitement the patient sank on the twenty-seventh day of his disease.

Dissection fourteen hours after death.—The lungs were so congested as to sink in water, and the incised surfaces exuded a large quantity of a frothy and bloody fluid. All the bronchial tubes were filled with a similar secretion. There were no tubercles. The pleuræ presented a small quantity of cadaveric effusion; but the pericardium contained about six ounces of a pale serum. The heart was of natural size: it was very flabby and softened, the left ventricle being more engaged than the right; the abdominal viscera were healthy.

This case is principally interesting as showing the signs of a weakened and softened heart in a case of unusual duration, and at a period when, in most instances, the organ has passed through the process of softening and recovery. No observation was to be had of the heart until after the twenty-first day; and it is probable that the softening process had existed long before. Its retrocession, perhaps, was delayed by some error in treatment. Had this patient been given stimulants at an early period, the result would, in all likelihood, have been different.

CASE LIV.—*Aggravated nervous symptoms in a Case of Petechial Typhus; Cessation of the first sound over the left side; Extreme softening confined to the left ventricle.*

This patient, a youth aged 19, first came under our observation on the eighth day of his fever. He had had delirium, epistaxis and dyspnœa, and the eruption appeared on the fifth day. On the ninth day he complained of a feeling of extreme cold. His skin was hot, and covered with a thick crop of petechiæ, of a bright red colour. He had delirium, watchfulness, rolling of the eyes, a dry and fissured tongue, and his lips were covered with sordes. There were no abdominal symptoms. Pulse 116, regular and full. The heart's impulse was vigorous, and both sounds distinct; at the apex the second predominated, while at the base the sounds were proportionate.

On the following day the nervous symptoms were aggravated: the skin was very hot, and the maculæ more abundant; pulse 132, weak and compressible. The sounds of the heart were very weak, and had the fœtal character. The impulse remained distinct under the sternum, but was wholly wanting below and to the left of the nipple. On the eleventh day delirium was incessant; and the subsultus tendinum affected not only the limbs and face, but the trunk itself, which was affected by convulsive shocks, often so severe as to throw the body into an arch, the patient resting only on his head and heels, yet the pulse had fallen to 121, it had more strength, and the impulse of the heart had increased. Still, though both sounds were audible over the right side, there was perceptible at the left but a single sound, and that the second. He died on the thirteenth day. The pulse had become imperceptible, although on the day before death there was a jerking impulse of the heart.

Dissection, twelve hours after Death.—The general muscular structure was found unaffected, firm and red, with the exception of the great pectorals, which were slightly softened. The lungs appeared healthy, but were congested posteriorly; the air tubes were full of frothy mucus, and the structures between the tracheal cartilages, and also the circular fibres of the second order of bronchial tubes, had a remarkably livid appearance. The pericardium contained a considerable quantity of serum. The size of the heart was natural, but the left ventricle had a livid colour, contrasting strongly with that of the right; it was also much softer; on its lateral surface were two spots still more livid, the colour being a decided purple; this change extended for about two lines into the substance of the ventricle, and the patch nearest to the apex was the larger and deeper of the two; the whole substance of the ventricle was much softer than natural, breaking down under the slightest pressure, yet the fleshy columns were not in the least altered, and the right ventricle was quite healthy. No disease was found in the large vessels or the abdominal viscera.

The principal interest of this case consists in the remarkable circumscription of the disease to the walls of the left ventricle. In fact the right ventricle seemed to have wholly escaped, and the physical signs were singularly in accordance with the results of dissection. In no other case have we observed so complete an absence of impulse at the left side, while at the right it was so distinctly observed; nor have we any other instance of so remarkable a contrast being found between the appearances of the two ventricles on opening the body. The immunity, too, of the carneæ columnæ is to be noted, especially when the extreme degree of softening of the ventricular wall is recollected. We have not hitherto attended with sufficient care to the comparative examination of the fleshy columns and the substance of the ventricle; but it may be stated generally that the softening process is less developed in them, for we often find that, while the ventricle breaks under the finger, these portions will bear considerable pressure; and Louis observes that, though the internal surface of the ventricle is often of a deep colour, yet this appears to arise from imbibition of blood rather than from the extension of the softening process. Is this comparative immunity of the fleshy columns to be regarded as one of those inscrutable provisions of nature by which she seeks to preserve the great functions of an organ even to the last possible moment? For we can easily understand how much more dangerous the typhoid softening of the heart would be if those structures which seem to govern the proper action of the valves were affected equally with the general mass of the heart.

Case LV.—*Petechial Fever, with intense Pulmonary Disease; Death on the twenty-second day; Enlargement and softening of the Heart.*

A man, aged 35, had been largely bled at the commencement of his fever. He was admitted on the twelfth day in a state of extreme collapse and prostration: pulse 120, very small and indistinct. The surface was cold, and the skin covered with a dark-coloured eruption, forming small patches. He had severe cough, with hurried and difficult respiration, and a large muco-crepitating râle existed over the posterior portion of the chest. The heart's impulse could scarcely be felt; and neither the first nor second sounds could be discovered. Some re-action took place on the fourteenth day: the face was flushed, the delirium high, and the maculæ more abundant; pulse 116, much stronger. Yet, the heart's impulse was not improved; the first sound was everywhere inaudible, but the second could be discovered by applying the stethoscope to the epigastrium. The obscurity of the sounds of the heart, however, might have arisen from the loudness of the râles in the lung; yet the fact of our hearing only the second sound in the epigastrium proved that the force of the systole was really diminished. On the sixteenth day the second sound, as heard in the epigastrium, was very distinct, but without any return of the first. He sank on the twenty-second day, *all the sounds of the heart having been absent for forty-eight hours previously.* It was remarkable that after the first day the pulse ranged between 104 and 110.

Dissection, eighteen hours after Death.—The pericardium contained a good deal of serum; and the whole heart was enlarged and very flabby; the left ventricle was much more softened than the right, and easily broke down under the finger. We were only permitted to examine the heart.

These cases, when compared one with another, present a remarkable similarity,—not only as to the general character, but the accompanying circumstances of the disease. We see in them all the special typhoid disease of the heart, as shown by the softening of the ventricles, without any evidence of inflammatory action.

In seven out of the eight cases the pericardium contained a quantity of serum greater than could be attributed to cadaveric effusion. It averaged, probably, at six ounces. The serum showed no traces of coagulated lymph. In one the state of the pericardium was not noted.

Congestion of the lungs, especially in their posterior portions, was noted in four cases; and in three at least of the remaining instances the physical signs and symptoms left no doubt that a similar condition existed.

In two the pectoral muscles showed a degree of softening.

Softening of both ventricles occurred in seven cases. In two the change predominated in the right; and in five in the left ventricle. In one the left ventricle alone was engaged. The state of the septum cordis is noted but in one case, in which it was found much softened, and in two it was observed that the fleshy columns retained their firmness.

The state of the abdominal viscera was not noted in two of the cases. In four no disease was found; the two remaining subjects presented softening and vascularity of the mucous membrane of the jejunum and ileum; and in one of these, ulceration of the last portion of the ileum was observed.

In none of the cases was the brain examined.

Finally, it was found that petechiæ in different forms, and undergoing various changes in the course of the disease, occurred in all the patients.

But the frequent co-existence of softening of the heart and a well-marked petechial eruption does not warrant the conclusion that there is any necessary connexion between the two affections. Cases are frequent in which, with the best-marked eruptions, we find the heart's action continuing vigorous up to the period of death. Of such, examples have been already given. I exhibited some years since to the Pathological Society the heart of a man of powerful development, who died on about the twelfth day of typhus, with large and dark-coloured maculæ. The first sound remained undiminished; in fact it predominated over the second throughout the entire case. The muscular structure of the heart was red and firm; the valves were healthy, and there was nothing abnormal in the heart except a long coagulum which passed from the right ventricle into the pulmonary artery. In this case no beneficial effect was obtained from the use of wine.

On the other hand, we have found, after a large experience, that the signs of debility, if not of softening of the heart, are by no means so frequent in those cases which writers term typhoid, as distinguished from typhus fevers. Without at present discussing the question as to the identity or the separate nature of typhus and typhoid fever, it may be safely said that the characteristic signs of softening of the heart are rarely met with in the non-petechial cases, or in those where but a few rose-coloured maculæ appear; and even when, in such instances, the heart shows signs of weakness, we find that the evidences of the organ going through a regular process of disease are by no means so well marked; yet it sometimes happens that, in these very cases, we must use stimulation boldly. A case of this kind is given in my original memoir. The patient, a woman advanced in life, had been three weeks ill previous to admission. We found her in a state of extreme prostration: the surface cold, and the action of the heart feeble and irregular; and it was not until after the eighth day of the exhibition of wine, and various other stimulants, in great quantities, that the state of the heart began to improve. Indeed, nothing but perseverance in the use of stimulants could have saved this patient. The form of fever under which she laboured is rarely seen in our hospitals, and is characterized by extreme adynamia, unaccompanied by the signs of "putrescence." It was of long duration, and apparently uncomplicated with visceral disease.

Cases, however, of non-petechial fever, presenting the signs of softening, may occasionally be met with. Of this condition the following is a good example.

CASE LVI.—*Non-maculated Fever, with slight abdominal and pulmonary irritation; Absence for several days of the first sound of the Heart; Second sound distinct with its own impulse; Diminished systolic Impulse; Thrilling Pulse; Recovery.*

A boy, aged 13, after having been exposed to contagion, was found on the seventh day in a state resembling inflammatory rather than typhoid fever. The carotid and temporal arteries were acting strongly, as was also the abdominal aorta. Pulse 116, full and bounding. He had thirst, a white tongue, and hot skin. There were no evidences of disease in the head, lungs, or abdomen.

The impulse of the heart was distinct, and had a certain thrill, and over the left cavities the second sound predominated. But this want of proportion appeared to arise from an augmentation of the second rather than from any deficiency of the first sound. Over the right side the second sound was not so loud as on the left.

On the eighth day the vascular excitement was lessened, and the pulse 108, and of good strength. The impulse was not so thrilling. At the nipple the predominance of the second sound was more obvious, at least at the left side. We observed the first sound to be weaker during inspiration than expiration. On the tenth day the first decided loss of the systolic sound and impulse at the left side was observed. The second sound remained remarkably well defined and attended with its own impulse. These phenomena were very evident over the left side.

On the twelfth day the signs of weakness of the heart were very evident, and yet the general symptoms were in no respect worse. At the base of the heart the first sound was wanting, but it appeared as the apex was approached. It could be feebly perceived under the sternum. When we examined the left cavities, while the patient was in the recumbent position, the second sound could be heard with great distinctness, while the first was hardly to be discovered. When he sat up the second sound was a shade more feeble, while the first continued as before.

The fourteenth day having arrived, we found that the ventricular impulse was reduced to the slightest trace, while that attendant on the second sound was extremely distinct. This second sound was clear, well defined, and so loud that it could be heard over the entire anterior portion of the chest, and yet it was not even so distinct as on the day previous. The first sound could scarcely be discovered anywhere; a trace of it existed towards the apex, and at the left side of the sternum; but it required a practised ear to discover it. The pulse had a thrilling hemorrhagic character. The want of the first sound was most remarkable; there was no bronchitis by which it could be obscured, and the patient's strength was but little, if at all, impaired. He could sit up or move about the ward without inconvenience or the slightest exhaustion. On the next day the thrill of the pulse was diminished, as was also the loudness of the second sound; but little change occurred up to the nineteenth day, when the general symptoms had much subsided, and the pulse was 82, and of good strength; the loudness of the second sound diminished, but the first was still completely absent; the second impulse continued perceptible, but the first was wanting. On the twentieth day the first impulse began to appear, and on the following day, the pulse having come down to 80, the double impulse was perceived; the second impulse being still stronger than the systolic. From this time the heart slowly recovered, but was scarcely restored to its natural condition, when the patient found himself sufficiently well to leave the hospital.

The extraordinary loudness and distinctness of the second sound, coincident with the strong and thrilling pulse, the extinction of the systolic impulse and almost of the sound, and the preservation of the second impulse, are circumstances of great interest; but it is not easy to determine the actual state of the heart in this case. That a weakened state of the left ventricle existed is obvious, but that this was not connected with general debility is manifest from the absence of all the characters of asthenia, and we can only attribute it to the occurrence of softening and weakening of the ventricles, or to the existence of nervous debility,—a semi-paralyzed condition of the heart. Of the existence of this latter condition we as yet know but little; we have, it is true, seen several instances of apparent debility of the heart, in which its speedy restoration, and other circumstances, led us to the belief that actual softening had not occurred, but we have seen few cases in which the extinction of the first sound or impulse continued for so great a length of time. We may then conclude that, in certain non-petechial fevers, the heart undergoes a process of softening and weakening, although the supervention of this disease, under such circumstances, must be looked upon as a rare occurrence.

The last illustration which I shall give of the depressing effect of typhus upon the heart is an instance of the temporary suspension of an old valvular murmur. Such an occurrence might be anticipated, for the same weakness of the ventricle which would cause a suspension of the first sound would also, if the mitral, and in some cases even the aortic, valves were the seat of a pre-existing murmur, cause its diminution or extinction. We know that, other things equal, the murmur from valvular disease is directly as the force of the heart, and that whatever diminishes the contractile power of the organ lessens the amount and alters the character of the valvular sound. Thus by rest, low diet, or the use of digitalis, we may sometimes, even in cases of extreme valvular disease, cause the murmur in a great measure to disappear: but this is only temporary, for the murmur is re-established when the force of the heart is restored.

Substituting, then, a pathological for an artificial cause, we find that in typhus fever, when it attacks a patient who has been the subject of old valvular disease, the weakness of the ventricular contraction may cause a suspension of the valvular sound. Of this occurrence, of which we have seen more than one instance, the following case is an example:—

CASE LVII.—*Maculated Fever; Disappearance of an old Cardiac Murmur, coincident with the signs of softening and weakness of the Heart.*

A woman, aged 25, was admitted in November, 1846, on the sixth day of fever. Seven years before, she had experienced several attacks of rheumatic disease, and was under the care of Dr. Graves for a consequent affection of the heart. This patient had been in the hospital three weeks previous to the attack of fever for a fresh access of rheumatism, and at that time a distinct bellows murmur accompanied the first sound of the heart, and this continued up to the time of her leaving hospital. On her second admission we found the trunk covered with large red maculæ; the tongue was dry; the eye suffused; and the pulse 120 and weak. When she took a deep inspiration a humid râle was heard in the posterior portions of both lungs, but particularly the right; the first sound of the heart was not well defined, but it could scarcely be said that there was murmur. On the following day the pulse was 126, and there was not the slightest trace of murmur. On the ninth day the pulse was very weak; the impulse of the heart was feeble; and the sounds had much of the fœtal character. But little change occurred up to the fourteenth day, at which time the bronchial râles were intense. From this time convalescence advanced, and the murmur was soon found to return. Her recovery from the fever was complete; but she left the hospital with a heart presenting precisely the same phenomena that it did on her admission.

After a period of six weeks this patient was seen by Dr. Heslop, then my clinical assistant, who found a loud and harsh murmur with the first sound.

DEVELOPMENT OF MURMUR OF THE HEART IN FEVER.

Having now examined the phenomena of the heart when under the influence of typhus in its aggravated forms, we may proceed to consider a class of cases in which the symptoms and signs are very different.

When, in 1837, I published my Observations on the state of the Heart in Typhus Fever, I had not observed the phenomena which are now to be described. It has been seen that failure of the systolic impulse and sounds, especially of the left ventricle, unattended with murmur, and occurring in cases of aggravated typhus, was the principal indication of the lesion of the heart. In the course of some years, however, a class of cases, of which isolated examples had not unfrequently occurred in our wards, became frequent, and were characterized by the following circumstances:—

1. The petechial eruption was generally wanting, or occurred in the form of a few scattered and pale maculæ.
2. The disease was of short duration, but very liable to relapse.
3. There was but little evidence of softening of the heart; and the use of stimulants was but seldom resorted to.
4. A bellows murmur, or, in some cases, a prolongation of the systolic sound, was common, especially in the relapse; but this did not result from carditis.

We may hence divide the cases with abnormal cardiac phenomena in fever into two groups: in the one we have severe maculated adynamic fever, with the signs, in a few cases, of non-inflammatory excitement, but in most instances, of failure of power of the ventricles; while in the other we observe, in cases of fever (which is often of the non-maculated type,—a short and often relapsing affection,) the development of a murmur with the first sound of the heart, while at the same time the signs of softening of the left ventricle are wanting.

CASE LVIII.—*Non-maculated Fever; Relapse, with a cardiac murmur attending the first sound, diminishing in a great degree in the erect position.*

A lad, aged 18, was admitted on the second of June, 1847, labouring under a very simple form of fever, unattended by maculæ. He was convalescent in a few days. On the thirteenth he had a relapse, and on the next day was so prostrated that wine and ammonia had to be exhibited. Mucous râles were heard in the right lung, and the epigastric and right hypochondriac regions were slightly tender. The morbid signs of the heart appeared on the following day, the third of his relapse. We found a bellows murmur coincident with and masking the first sound of the heart. This was more evident over the left than the right side, *and when the patient sat up it diminished so as to be scarcely perceptible.* In four days his convalescence may be said to have commenced: the impulse of the heart became stronger. The rhythm, which on the fourth day resembled that of the fœtal heart, became natural, and in a few days all murmur disappeared, leaving the sounds of the heart with their normal character.

At nearly the same time during which this patient was in hospital another case of the development of this murmur occurred. The general condition and history of the patient were, however, different. A boy, aged 15, had suffered a few days before admission from an attack of measles. When first seen the eruption had nearly disappeared, but there was still a good deal of irritation in the chest and abdomen. He was pallid, and had an anxious expression, and a soft murmur (apparently mitral) existed with the first sound. His convalescence was slow, but he finally recovered, and the murmur wholly disappeared, although no special treatment had been directed to the heart.

CASE LIX.—*Simple non-maculated Fever in an anæmic Subject; Relapse; Development of murmur with the systolic sound.*

A woman, aged 24, who had suffered from suppression of the catamenia and leucorrhœa, went through the mildest form of non-maculated fever, which lasted but a few days. She had a relapse before leaving the hospital, and, as often happens, the symptoms in the relapse were much more severe than in the primary attack. She had extreme prostration, and much irritation of the lungs. The face was pale. On the third or fourth day we discovered a systolic murmur at the left side, most evident at the base of the heart. The impulse had a good deal of force. With a view to the relief of the bronchitis, we applied a blister to the chest, and in twenty-four hours the murmur disappeared. No observation was made as to the effect of change of position.

For some time after these cases were observed we had a great number of examples of these relapsing fevers in our wards. A considerable proportion of them occurred in patients under the age of 25, and the following circumstances were more or less common to them all:—

1. The first attack of fever generally lasted about five days, but in some its duration was even less. The fever was of a mild type, and maculæ were not present.

2. After an interval of from five to ten days, the patients relapsed, and the symptoms were then often aggravated. There was greater prostration, and the signs of mucous irritation were much more prominent.

3. In a few cases there were as many as three, and in a still smaller number even four, attacks of fever (including the primary seizure.) We did not find that the exhibition of bark in the apyrexial state had any beneficial effect.

4. In most of these cases we found that the signs of softening of the heart were wanting; but the systolic sound, especially in the relapse, was prolonged, and in many instances a well-marked bellows murmur was established. In a few a murmur could be heard in the course of the carotids, but none of the accompanying signs or symptoms of carditis were present.

5. Enlargement of the spleen, though not to a great extent, was often found in the second or third relapse.

6. The murmur was in some cases but a prolongation of the first sound; but in others there was a distinct bellows murmur. This we observed in several instances to be most evident in the recumbent position, and nearly wanting when the patient sat up. It subsided during the final convalescence, but its duration varied in different cases.

This murmur, whatever may have been its seat and actual cause, is clearly to be placed in the category of inorganic murmurs. And its frequent development in the relapse, in cases too where enlargement of the spleen was observed,—while at the same time the signs of softening of the ventricle were wanting,—makes a strong case in favour of its being in some way connected with a depraved state of the blood. Further, it appears probable that, notwithstanding the passage, as it were, of one sign into the other, there were in some of these cases two morbid sounds,—one, the mere prolongation of the systolic sound, the other, a true murmur, having its seat in a valvular orifice. Of these, the first was the most frequent; and a corresponding condition of the first or systolic impulse, in which it had a protracted and somewhat vermicular character, was occasionally observed. The prolongation of the systolic sound is not uncommon in cases, not only of this form of disease, but also in other febrile affections. We have often found it after measles, when the disease was of an asthenic character; and again in cases of typhoid variola. It doubtless may be met with in many other affections attended with special forms of fever.

A few more instances of these conditions may now be studied, after which we shall examine the rare occurrence of murmur in cases of maculated fever.

CASE LX.—*Simple Non-maculated Fever; Prolongation of the first sound, afterwards passing into a bellows murmur, developed on the seventh day.*

A girl, aged 12, was attacked six days before admission with the usual symptoms of the then epidemic non-maculated fever. She had thirst, heat of skin, quickened pulse, and general pains. The heart was acting with sufficient strength, but there was a decided prolongation of the first sound. On the ninth day the pulse rose from 88 to 100, and the febrile symptoms were increased. The heart acted with considerable force, and the prolongation of the systolic sound amounted to a decided bellows murmur, which was most evident between the nipple and the left border of the sternum. It could be traced upwards along the aorta, and downwards towards the apex, but in both directions it progressively diminished, and on the extreme left of the chest it could scarcely be said to exist.

Little change occurred until the eleventh day, when it was found that between the nipple and sternum the first sound, and with it the bellows murmur, had disappeared. The first sound was slightly audible behind the sternum. Stimulants were ordered, and in the course of an hour the action of the heart had greatly improved. On the twelfth day the first sound had returned, and with it the bellows murmur. Pulse 92; the tongue loaded and dry, and the teeth covered with sordes. In two days the pulse came down to 80; convalescence went steadily on; and in the course of a week the murmur, which had been gradually declining, and which, eight days before, had been heard along the course of the aorta, was but feebly audible at a spot between the nipple and sternum. In a few days she left the hospital in good health, all murmur having disappeared from the heart.

In this case it was repeatedly observed by Dr. Heslop, that the murmur disappeared whenever the patient was made to sit up. The case may be taken as a good example of the occurrence of these signs in a fever which, though of longer duration, and not having the relapsing character, was clearly of the same nature as that which has been already described. With respect to the failure of the first sound of the heart, and with it of the murmur, we may hold, that it was caused by temporary weakness of the organ, unattended with the state of softening which accompanied the maculated cases. It will be remembered that in a very short time after the use of wine, the systolic sound was restored. The dependence of a clearly inorganic murmur on the dynamic condition of the heart is a fact worthy of notice.

Case LXI.—*Prolonged first sound, passing into Cardiac Murmur, existing both in the primary Fever and in the Relapse.*

A lad, aged 17, very pale, and whose general aspect was similar to that of a chlorotic female, was admitted on the fourth day of fever, complaining of pain, thirst, and headach: speech incoherent; delirium in the night; pulse 120; perspiration abundant. Dr. Lees observed that the left hypochondrium was extensively dull; the heart acted with considerable force; and the first sound was distinctly prolonged. This prolongation of sound was most evident between the nipple and the left border of the sternum, and a continuous musical murmur was heard at the right side of the neck. In twenty-four hours the pulse fell to 64, and a copious perspiration greatly relieved him. This was not wholly critical, for his convalescence advanced slowly for several days, when he relapsed. During the relapse the prominent symptoms were prostration of strength, and extreme pallor. He finally left the hospital in a very anæmic condition. The murmur was perfectly distinguishable when he was lying down, but either quite inaudible or much diminished in the erect position.

Case LXII.—*Relapsing Fever, with prolongation of the first sound.*

A man, aged 30, was admitted in November, 1847, and went through his fever with but little excitement of the pulse. A certain degree of jaundiced tint existed over the body. On the sixth day he was free from fever, and in four days his pulse fell to 44. Five days having passed, he relapsed, with rigors and pains in the limbs; pulse 80; the first sound of the heart was prolonged, and change of position failed to affect the sign. This prolonged murmur was loudest in the usual place, and could be feebly heard to the right of the sternum. During the next two days it diminished, and was absent in the upright position; and on the third, or perhaps the fourth day of the relapse, the murmur altogether subsided.

Case LXIII.—*Fever, with Cardiac Murmur in the Relapse.*

A girl, aged 22, admitted in November, 1847, went through a short and mild fever. She had a venous murmur in the neck, but no murmur whatever in the heart. After a few days she relapsed, with shivering, heat of skin, and pulse 120. The left hypochondrium was tender, and its dulness seemed increased. The first sound of the heart had a distinct murmur. A copious perspiration occurred in about three days from the period of the relapse, and in the course of a week she was free from fever. During this time, however, an evident murmur existed with the first sound, but completely disappeared in the upright position. In the last report of this case the murmur is mentioned as disappearing. This patient was labouring under amenorrhœa.

Let us now examine two cases of the short fever, in which cardiac murmur from organic disease was discovered. In both the result was fatal, and we had the opportunity of making post-mortem examinations.

A girl, aged 19, was admitted, under the care of Dr. Lees. Her symptoms had been of three days' standing, and were contracted during attendance upon her mother, who had been shortly before admitted into our wards, with the symptoms of what was known by the name of the short fever. She had debility, and want of sleep, her countenance was pale and anxious, and a circumscribed flush existed in the centre of both cheeks. Pulse 130, small and feeble; respirations 30, and tongue white. The heart acted with some force, and the first sound was attended with a rough murmur, which was loud under the mamma, and towards the apex, but feebly audible under the sternum. Dr. Lees came to the conclusion that this murmur proceeded from some organic disease of the heart. The patient died on the seventh day of her illness. The left auricle was found enormously dilated and hypertrophied; the left ventricle also was hypertrophied; the mitral valves were fixed together by their borders, and thickened down to their bases. Viewed from the auricular side the orifice presented the appearance of a cartilaginous infundibulum, the apex of which looked towards the cavity of the left ventricle. The orifice obviously admitted of regurgitation: there was no evidence of acute endocarditis. The pericardium was dotted over with minute red specks. It was ascertained that this girl, for some months previous to her fatal illness, had suffered from an attack of rheumatism, or rheumatic fever.

CASE LXIV.—*Cardiac murmur; Albuminuria; Amenorrhœa; Death.*

A girl, aged 22, was admitted in October, 1847. She complained of pains in the feet, with swelling and a sensation of cold after being in the erect position. Her appearance was anæmic. She was emaciated, and a well-marked venous murmur was detected in the left side of her neck. After the exhibition of tonics she slightly improved, but she continued to have a very wretched appearance. Nothing remarkable was observed in the sounds of the heart except a certain degree of harshness, apparently indicative of great excitability of the organ. In about a fortnight after admission she was attacked with severe vomiting, followed by a state of extreme collapse. The heart was found to act with great force, although the pulse was miserably weak; and below the mamma a loud rasping murmur attended the first sound. Thenceforward she became progressively worse; the action of the heart continued excessive, with a loud rasping sound. She had extreme prostration, and the urine was albuminous, and of low specific gravity. On the fifth day of the attack œdema of the lids of the right eye appeared, and the conjunctiva was raised from the sclerotic by serous effusion. This increased until the eye was closed; the œdema spread to the scalp; and she died on the tenth day from the commencement of the attack.

Dissection, thirty hours after Death.—The body was much emaciated, and there was very great œdema of the lids of the right eye, and of the corresponding side of the head. No œdema existed in any other part of the body. The right kidney was small and flabby, and a large portion of its substance was degenerated into a yellow mass, extending deeply from its anterior surface. The whole organ was very pale, and yellow fatty-looking masses existed between the tubuli, which, towards the centre, and at one end, were extremely atrophied. In the cortical portion, and on the surface of the kidney, little bloody points could be seen. There were no abnormal adhesions of the supra-renal capsule. The left kidney was much increased in size; it was flabby, and contained a small cyst. The liver was soft and pale; the lungs and pleuræ healthy. About six ounces of bloody serum were found in the pericardium, but its surface was healthy. The heart, viewed from the pericardium, had a dark colour; it was extremely flaccid, and all its veins much distended. We found the left ventricle thicker and of somewhat greater capacity than natural. The mitral valves were adherent, so that the orifice was so much diminished that the tip of the little finger could not be passed through it. A small portion of lymph hung from near the point of the anterior valve. On looking at the orifice, at its auricular side, nearly two-thirds of its extent were seen to be surrounded by vegetations of reddish lymph in considerable quantity, so that the valves appeared to be incapable of closing the orifice. The lining membrane of the left auricle was of very dark colour; there was some thickening of the aortic valves; on one of them a slight deposit was observed; no disease existed in the right side of the heart.

Although differing in their nature, the last two cases are worthy of study, as illustrative of some of the difficulties which attend the subject in hand. In both cardiac murmurs existed. The patients entered hospital at the very period when we had so many cases of the short fever with the development of cardiac murmur; and one of them actually laboured under the epidemic disease. This was in Case LXIII.; and many who saw the patient came to the conclusion that the murmur was not organic. Dr. Lees, however, formed a more correct opinion, founding his diagnosis on the unusual character of the murmur, the force of the left ventricle, and the fact that the upright position produced little or no change in the sound. The patient had, doubtless, long laboured under this affection; and it is probable that the impediment to the circulation assisted in causing the fatal result.

In the second case a different set of symptoms and signs existed; and there was less difficulty in the diagnosis. When the patient was admitted there was nothing remarkable found in the action of the heart, except that kind of harshness of sound which we often find in excitable hearts. But there were neither the signs of typhoid softening, nor the murmur observed in the short relapsing fever which was then epidemic. In a fortnight an attack of vomiting, followed by general prostration, and great excitement of the heart, took place, and then the loud rasping murmur was established.

Here was a combination of circumstances which, as inconsistent with anything which we had observed in fever without inflammatory disease, could only be held to indicate an endocarditis occurring in a patient already anæmic. With reference to physical diagnosis simply, it may be remarked, that a rasping murmur *with excitement of the heart*, was never found by us, either in typhus, or in the relapsing fever with cardiac murmur.

Having now examined three important groups of cases,—namely, maculated typhus, with excitement of the heart; maculated typhus, with weakness and softening, unattended by murmur; non-maculated fever, without softening of the heart, but with the production of a murmur at some period, we may investigate a fourth class, in which, with maculated fever, a bellows murmur occurs in the heart. So far as our knowledge goes, such cases are very rare; a circumstance difficult of explanation, especially when we recollect the frequency of the non-inflammatory murmurs in the short relapsing fever.

CASE LXV.—*Maculated Typhus, with signs of weakness of the Heart; Musical murmur accompanying the first sound.*

A miller, aged 45, was admitted under the care of Dr. Lees, in August, 1847. He had then been four days ill, and the fever had not been modified by any improper interference. On the fifth day the maculæ were red and evident over various parts of the trunk; the tongue was very dry and brown, the skin dry, and the patient's thirst was great. Pulse 96; the first sound very weak, especially at the left side of the heart, where it was scarcely audible. Some congestive râles existed at the base of both lungs. *On the eighth day a distinct musical murmur was observed with the first sound of the heart*, and the pulse had fallen to 80. On the 10th the pulse was 72; the heart very weak, so that the impulse could scarcely be observed; the first sound was greatly diminished, and sometimes appeared like a *souffle*, without any of the true systolic sound. On the following day all murmur had disappeared, but the first sound, particularly just above the nipple, was extremely weak. This was on the 31st of August, but we were surprised to find, on the next day, that the musical murmur had returned, and the first sound, in fact, was almost replaced by it. This murmur was most intense at a spot immediately below and to the left of the nipple. Between the nipple and the left border of the sternum it was also audible, but here it had lost the musical character. It was not audible behind the sternum or above the nipple. On making the patient sit up, the musical character of the murmur completely disappeared, the effort inducing a momentary excitement of the heart. On continuing the examination it became evident that a *souffle* still existed at the spot where the musical murmur had been audible, but everywhere else all murmur had vanished, and no disproportion could be observed between the sounds. These observations were repeated, and with the same result. On the right side of the neck a continuous venous murmur was to be heard. From this time his convalescence was progressive. The murmur lost its musical character, but could be heard as a simple *souffle* at the apex, and between the nipple and sternum. In the latter situation it disappeared when the patient sat up, and at the apex it was much diminished. Soon after, he left the hospital, but returned in a month to see his daughter, who was then labouring under non-maculated fever. A careful examination was made of his heart. When the patient was in the recumbent position, the slightest murmur attended the first sound at the apex, while between the nipple and the sternum there was nothing but a trivial prolongation of the systolic sound. Another examination was made a month subsequently. A slight prolongation of the first sound still continued, perhaps more audible when he lay down. The pulse was tranquil, and the impulse of the heart rather strong.

The phenomena of this case were repeatedly and accurately observed by my colleague and myself. All the facts connected with the murmur point to the conclusion that it was inorganic. The co-existence of the venous murmur in the neck; the varying character of the musical sound, which latter was most evident when the heart was weakened; and its modification by position, strengthen this conclusion. On the other hand, the continuance of the prolongation of the first sound for such a length of time after convalescence,—when also the action of the heart was vigorous,—is to be taken into account; and it is to be noted, that although the patient's health was excellent, he stated, on being interrogated, that he had felt some increased action of the heart ever since his fever. Yet I believe that this patient had no valvular affection. I have repeatedly observed, not only in fever, but in other adynamic diseases, that the prolongation of the first sound may continue for an indefinite period after convalescence. This is a special condition which has not been sufficiently attended to. Had this patient been the subject of valvular disease produced during his fever, the signs would have become more manifest as the heart regained its natural force.

In the next case we have an example of murmur developed during the convalescence from maculated typhus.

CASE LXVI.—*Murmur with the first sound of the Heart, observed on the twenty-first day, in a Case of Maculated Fever.*

A man who, as stated by his friends, had been seven days in fever, was admitted on the 31st of August, 1847. He passed a restless night, sitting up in his bed, and crying out. This was succeeded by stupor. The eyes were injected, the tongue furred, and the skin hot, dry, and covered with dark-coloured maculæ. Pupils dilated; pulse 140. The ileo-cœcal region was tender, and gave distinct gurgling on pressure. The heart was acting with great force, and the sounds were proportionate. These unfavourable symptoms, however, soon began to disappear. The pulse came down gradually, and on the thirteenth day was only 88. On the sixteenth the maculæ had almost disappeared; his tongue was nearly clean, and his sleep restored. In two days more the pulse was 76, and a few sudamina appeared on the abdomen.

On the twenty-first day a loud venous murmur was heard on the right side of the neck. The action of the heart was slightly irregular, and with an obscure murmur between the nipple and sternum. This ceased in the erect position. There was extended dulness over the region of the spleen. In this state he continued for a week. He was pallid, and progressed slowly. He was then transferred to a convalescent ward, in which were two patients who had recovered from variola, when, in a few days, he was attacked with that disease in a mild form. He finally left the hospital in good health.

A certain degree of prolongation of the first sound, occurring in cases of ventricular softening, and after the impulse was restored, has been already noticed; and it may be stated generally, that this condition, as met with during the convalescence of typhus, is rarely found to amount to an actual murmur resembling that so frequent in the relapse cases of the short and non-maculated fever. It is also to be met with during convalescence in other diseases. We have lately observed it in two patients, who recovered from a bad form of variola, and whose prostration necessitated the free use of wine from an early period. Doubtless the same phenomenon occurs in many of the acute yet adynamic diseases. Like the murmur in the short fevers, this prolongation of the first sound disappears or becomes diminished in the erect position, but perhaps in a lesser degree. It is, however, not always affected by position.

APPLICATION OF THE PRECEDING OBSERVATIONS TO THE TREATMENT OF FEVER AND OTHER DISEASES.

We would take but a limited view of the importance of the facts now stated, if we considered them only in relation to the diagnosis of structural or functional derangements of the heart in fever. But these investigations afford not only a series of novel pathological facts, but furnish us with new instruments in the treatment of disease. Further, they have assisted in confirming the doctrine, that in a large proportion of the acute and febrile diseases, local affections arise which are not only non-inflammatory, but to use the words of Louis, of a nature the very opposite to inflammation. It may be truly said, that when this great truth in its extended application has been engraved upon our minds, we have largely advanced in our knowledge of true medicine; and that, on the other hand, the practitioner who is ignorant of, or wilfully shuts his eyes to the number, variety, and importance of the acute but non-inflammatory affections, is unfit to deal with disease.[1]

[1] We may regard every new fact in pathology, or pathological anatomy, without exception, as either immediately fruitful in its application to practical medicine,

There are many cases besides fever in which the determination of the state of the heart as to its contractile force is of practical importance. Let us enumerate some of the acute diseases in which careful examination of the heart furnishes important indications in the treatment of the general malady:—

1. Typhus fever with maculæ.
2. Typhus fever without maculæ.
3. Protracted adynamic fever.
4. Non-maculated fever.
5. Variola with typhoid symptoms.
6. The low forms of scarlatina.
7. Delirium tremens complicated with low fever and with affections of the great viscera.
8. Typhoid erysipelas.
9. Asthenic or typhoid pleurisy.
10. Typhoid pneumonia.

or as something that at a future time will show its value in this direction, and this should be a great encouragement when the results of our labours appear only as additions to a catalogue of facts Notwithstanding all that has been done in that branch of pathological medicine which treats of the local affections secondary to essential disease, many practitioners consider them as original inflammations, and to this is to be attributed a large proportion of the errors in diagnosis and practice, so much to be deplored at home and abroad. If we inquire why it is that so many do not receive in its full extent the doctrine that a vast number of acute diseases cannot be explained by the theory of primary inflammation,—and again, that when inflammatory action does occur in them, it is of a re-active, secondary character, acting on tissues already altered by a process of a different kind, which is itself subsidiary to a general and essential condition,—we find that they are manifold. The opinions to be overcome are the growth of many years, and may be fairly dated from the period when accurate anatomical investigation was applied to elucidate diseased structure. As might be expected, an almost exclusive attention was directed to those manifest changes induced by inflammation, so striking not only in their earlier stages, but also in the successive periods of the process. And thus the doctrine of solidism, while it replaced the humoral theory, came to mean more than its name would imply, for while it referred diseases to an alteration of the solids, it taught that these changes had a common character dependent on some stage or phase of the inflammatory process. Alterations unattended with increased vascularity, tumour, or ulcerative action, were naturally overlooked, and this the more readily when no symptom of pain or disturbed function had been observed during life. Thus the facility which presented itself from the multiplication of instances of inflammatory action in all the great organs, and of connecting symptoms with some stage of these changes, paved the way to the doctrine of Broussais, which referred so many diseases, both general and local,

11. Asthenic pericarditis and endocarditis.

12. The acute pyogenic conditions of the system, affecting especially the joints and viscera.

Many other cases might be added to this list, but it is sufficiently extensive to show the class of diseases in which it is of great importance to ascertain the dynamic state of the heart from day to day.

It is not to be held, that in all the cases now specified there exists a true secondary softening of the heart. This condition, at least when arising under the laws which govern the secondary and periodic local diseases, has hitherto appeared to be peculiar to typhus fever in its well-marked cases; nor is it to be supposed that in the diseases now enumerated, which, though not typhus,

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to irritation or inflammation. Naturally captivating from its being, as it were, the first fruit of so much past labour in a new direction,—specious in its application to a general theory of medicine, and supported by the bold writing and great experience of its author,—this doctrine soon became on the Continent an accepted medical creed. In the British Isles, too, though more cautiously received, it influenced the minds of students, and of the younger members of the profession. Yet it must be admitted that a greater indisposition to receive the doctrine was manifested in these countries than in other parts of Europe. For with us conversion is a slow process, and conviction once established is not surrendered on light grounds, and the British physicians had already learned the value of a philosophical eclecticism. Still, as we have already observed, the doctrine of the inflammatory origin of many diseases became too popular with us. The writings of Hunter had a great effect in this direction, for his work was the text-book for the surgical student especially, whose mind was in the first instance directed to the study of inflammation, and little else. The unhappy division of the profession into separate corporations of Medicine and Surgery operated strongly in extending the adoption of an inflammatory theory of disease, for in the wards of a surgical hospital the surgical student saw little or nothing but the effects of inflammation. Thousands of practitioners were created who had never seen a case of typhus fever, and who, even when other forms of adynamic diseases presented themselves, were misled by the specious terms of diffuse or erysipelatous inflammation. It is difficult to change the opinions in which we have been brought up, and it often happens, especially in medicine, that when an opportunity is given of extended experience, the mind, from age, habit, or indolence, has become unfit to profit by that opportunity. When we reflect on this, and that for many years a large proportion of British practitioners have received an education almost wholly surgical, and then have entered on practice with but scanty knowledge of the non-surgical and the essential diseases in this country, still less of those met with over the wide surface of the British dominions,—we can understand how extensive must have been the application of antiphlogistic treatment to disease which was itself antiphlogistic.

have a typhoid character, the heart is necessarily in a weakened condition, for in them, as in typhus, we occasionally meet an excited condition of the organ, even when the general state is adynamic. But a large proportion of these cases present, in common with typhus, the signs of debility of the heart; and it may be laid down, generally, that these signs are more transitory and variable than in true typhus; and that the use of stimulants is less efficacious, and requires more caution, than in cases of typhus in which the signs of softening, as well as of debility, are present. In malignant scarlatina, in putrid variola, in the complicated and typhoid delirium tremens, notwithstanding the existence of great prostration and weakness of the circulation, it too often happens that stimulants fail to remove the typhoid state. We cannot say why this should be the case, nor can we as yet connect the failure of stimulants in the one set of cases, and their success in another, with the non-existence or existence of softening of the heart; but the fact appears established by clinical experience. Still it may be laid down that, even in the adynamic diseases which are not true typhus, the observation of the state of the heart is of great value to the practical physician.

The most important result of these investigations is, that when we find a weakened heart in a case of typhus, we may conclude, not only that stimulants are indicated, but that they will be borne well by the patient, and, in all probability, act in the most beneficial manner. And this observation applies to all those cases in which at least the signs of debility do not vary within short periods of time, that is to say, when they do not, as in some unfavourable examples, alternate with those of an excited heart. In the progressive failure of the impulse,—in the diminution and extinction of the systolic sound,—in that condition of the heart in which its sounds resemble those of the fœtus in utero, we have distinct and easily ascertainable indications for the use of wine, and an assurance that, in most cases, the remedy will be successful.

But, further, there is reason to believe that the more prominent and dangerous symptoms of adynamia in typhus, are often, as it were, foreshadowed by the early appearance of physical signs of a weakened and softened ventricle, and hence, by the timely observation of the heart's action, we can anticipate the general prostration, by freely resorting to wine even when the pulse is strong, the thirst urgent, the skin hot, and the maculæ of a vivid red colour.

We have seen, however, that the depressed condition of the heart does not occur in every case of typhus fever; that in some throughout the case its action presents nothing abnormal excepting the deviations from the ordinary rate; that in others, there is an excited state often co-existing with a weak pulse, and general signs of extreme adynamia; while in a third class we observe the progressive loss of power of the ventricles, advancing to a certain point at which the heart commences to regain its natural force. In the first of these classes wine is sometimes unnecessary; in the second its use is generally inefficacious; but in the third it becomes the sheet-anchor of treatment. And there is nothing more singular than the power which is given to the patient of bearing large quantities of diffusible stimuli without experiencing any of those effects on the nervous system that are produced in a state of health, when an unusual quantity of wine is taken.

It is not saying too much to assert, that our researches on the condition of the heart in fever have given a great facility in judging as to the necessity of stimulants in any given case of that disease, and that they not only furnish a rule by which the junior and inexperienced man may be guided, but give to every practitioner a greater degree of confidence in himself when he has to determine on the use, the increase, or the diminution of stimulants in fever. And, independent of all this, they have furnished us with new prognostics of great value. With very few exceptions it may be laid down, that the return of the impulse and of the first sound, under the use of stimulants, justifies a good prognosis, more especially if the rate of the pulse is falling; and conversely we find that the existence of an excited state of the heart, with a strong impulse, and clear and proportionate sounds, indicates danger, and the more so if the pulse be weak and rapid, and that its rate increases, rather than diminishes, under the use of stimulants.

We are not in a position to declare why it is that stimulants are not only so useful in the cases with softened or weakened hearts, but that their exhibition can be pushed so far, not alone without detriment, but with such singular advantage. We might inquire whether in these cases the brain is really in a condition analogous to that of the heart, or whether, owing to the deficient power of the left ventricle, it is temporarily anæmic, and suffering from the consequences of that condition. I have already, in speaking of the use of wine in the fatty disease of the heart, suggested that in the importance of stimulants, and again in the power of bearing them in large quantities, some analogy might be seen between cases of chronic weakness of the heart and those of the recently produced and transitory debility which occurs in fever. On the other hand, it is true, that so far as the occurrence of syncope in the erect position is concerned, we have observed this symptom but rarely in typhus with signs of the softened heart. For obvious reasons, however, the investigations on this point are deficient, and in the cases where syncope was observed, the altered position of the patient was the result of accident. And it may be that, owing to the operation of some of those singular conservative actions which are seen in acute disease, and especially in the class presenting the signs of periodicity, the brain in fever is enabled to bear the consequences of a weakened heart better than in cases of fatty degeneration. In the latter it commonly happens that we have not only weakness, but great retardation of pulse; and it will be seen that the pulse in fever was generally rapid during the different stages of the typhoid process in the heart, and that the slowness of action, such as is seen in the fatty heart, was ephemeral, and did not occur until the returning impulse and first sound had shown that the organ had recovered its force, at least to a great degree.

In connexion with this subject the researches of Dr. Hudson must be studied.[1] In a memoir published in 1842, Dr. Hudson after expressing his conviction of the value of the rule which I had announced, namely, that in the diminished impulse, and in the feebleness or extinction of the first sound, we have a new, direct, and important indication for the use of wine in fever, observes, that the indications with respect to the use of wine, derived from the signs of a weakened heart, are of equal value with reference to that of opium, that is to say, that opium may be used advantageously in cases of fever with delirium which show the signs of a weakened heart. And again, that the remedy will not answer when, with similar or nearly similar cerebral symptoms, the heart is acting with force. Comparing the cerebral symptoms in fever with those of delirium tremens, in which two essentially opposite states of the brain may occur,—one requiring stimulants and opium, the other bleeding and purging, Dr. Hudson comes to the conclusion, that similar conditions arise in fever, and that they depend in both diseases on the state of the heart:—the weak heart attended with symptoms of cerebral disturbance, which are to be combated by wine and opium; the vigorous heart, on the contrary, indicating that an opposite plan is to be pursued. But putting aside the question as to the use of opium, or its influence in that state of the brain which, it may be presumed, attends the weakness of the heart in fever, we find in Dr. Hudson's memoir some important points in which the investigations conducted at the Meath Hospital are deficient. The condition of the brain was noticed in four cases. In the first, an example of maculated fever, in which the signs of softening of the heart occurred, the veins of the pia mater were found congested, and the ventricles contained an unusual quantity of serum. The left ventricle of the heart presented a mottled appearance, all traces of muscular fibre being lost in certain portions which, varying in colour, closely resembled patches of hepatized lung in the different stages of pneumonia. In the second case muttering delirium and jactitation occurred, with loss of impulse and first sound. Death took place on the seventeenth day, and the brain and heart exhibited the same appearances as in the last case. The signs of debility of the heart occurred in the two next cases, and the organ was softened; in one of them the muscular tissue presented the unusual appearance of being infiltrated with blood. The brain was not examined in these two instances.

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[1] Observations on the Connexion between Delirium and Certain States of the Heart in Fever. With Cases. By Alfred Hudson, M. D. (Dublin Journal of Medical Science, First Series, vol. xx.)

But additional observations are wanting to establish the doctrine that in fever, with a weak heart, the brain is anæmic, so far as its arterial supply is concerned. Yet to this conclusion, all our observed facts, and the first two dissections recorded by Dr. Hudson, appear to point. Dr. Hudson's seventh case, however, is one which shows the necessity of a more extended investigation. A man was admitted on the sixth day of fever. He was maculated, and had low delirium, with a tendency to stupor. The extremities were cold, and the pulse 100, and weak. He was unable to put out his tongue; the heart's impulse was strong and visible, and both sounds could be heard. The head was shaved, and a cold wash applied. Eight leeches were placed behind the ear, and the back of his neck was blistered. The patient also had two grains of calomel exhibited twice a day. On the next day, the seventh of his illness, the prostration was great. He had muttering delirium, difficulty in swallowing, and decubitus on the back. The pulse was 112, the heart greatly reduced in strength, and the first sound absent on the left side. The calomel was continued, the patient was ordered four ounces of wine, and on the following day the heart's impulse was stronger, and the sounds were distinct. The pulse had risen from 112 to 120; it was weak. On the ninth day of his illness a fit of convulsions occurred, which lasted for three hours. The fit subsided after the loss of five ounces of blood from the temporal artery. The heart was acting violently, but the pulse could scarcely be felt at the wrist. He died on the following day, the heart's impulse having continued strong even while the patient was obviously sinking. On examination of the brain the convolutions appeared slightly flattened, and their surfaces were red and minutely injected. A large quantity of serum was found in the ventricles. The heart was firmly contracted, and empty.

We have here an exemplification of the principle already laid down, that in maculated fever with a feeble pulse, while the action of the heart is vehement, the prognosis should be unfavourable. The excited state of the heart, combined with the fact that the muscular structure of the organ was found firm and healthy, and also the occurrence of a severe and long-continued convulsion, presents a strong contrast between this case and one of softening and debility of the heart.

The loss of power of the heart, which occurred on the seventh day, must be considered as proving, that in the course of typhus fever a sudden, though temporary, debility of the organ may arise.

To this we have already alluded when speaking of the variableness which attends the secondary diseases of typhus.[1]

The following important case is given by Dr. Hudson. A girl, aged 16, was admitted on the twelfth day of fever, and went on well until the evening of the nineteenth day, when she became delirious; the heart's action was strong, and could be felt over the whole cardiac region; the delirium continued; the face became contracted and pale, and the extremities cold. The pulse was weak and thready, but the heart's impulse was strong and jerking. She died on the twenty-third day. The surface of the brain was found slightly injected; the arachnoid was opaque, thickened in patches, and with considerable sub-arachnoid effusion. The substance of the brain was unusually firm, and extremely vascular, and the large arteries at the base of the brain were loaded with blood; the lungs were healthy. The heart was found contracted and firm, and in every respect free from disease.

Other examples are given by this author, in which an active heart coincided with symptoms of cerebral irritation; but as in these cases there was no dissection we shall not further allude to them. It is very important, however, to find, that even in two cases, a condition of the brain was discovered, which would confirm the precept advanced in 1839, that the stimulating treatment in fever was less advantageous when there was permanent excitement of the heart. And the value of determining the condition of the heart is still more manifest when it is remembered how difficult it is to determine the existence of cerebritis in a case of typhus fever, and how general is the application of the great practical rule, that symptoms which are diagnostic of inflammation in cases which are not typhus lose their value to a great degree when they arise in the course of a maculated fever.[2] I showed in my original essay that, so far as wine was concerned, it was most successful in the cases with feeble hearts, and that it frequently failed in those of an opposite description; and with respect to opium, the researches of Dr. Hudson have made it probable that the same rule will apply, or at least we may conclude that the remedy will best agree with that state of the brain which is the opposite of active hyperæmia. The general symptoms which should guide us in the use of opium in fever were long since pointed out by Dr. Graves, but the application of our knowledge of the state of the heart to this question is due to Dr. Hudson.

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[1] In his comments on a case in which a feeble action of the heart succeeded to a state of excitement, Dr. Hudson has the following observations:—"That this deficiency of muscular power in the heart is very frequently connected with softening of its structure appears evident, but I much doubt that any such change is absolutely necessary to its production, since I have occasionally observed its signs during a short period of cases, which, after death, were found to have had a firm and healthy heart, while in the others, the signs have been totally wanting at times during life, and the heart has yet been found softened."—*Op. Cit.*

[2] See Louis, Recherches sur la Gastro-Enterite; Art. "Symptomes Cérébraux;"

Finally, in the cases of the musical murmur developed in typhus, and again in that more extensive class of non-musical bellows murmurs occurring in the short and so-called typhoid fevers, we may fairly hold, that the murmurs are of a nature very similar to those in ordinary cases of anæmia; and that they are to be attributed less to softening of the heart than to some change in the physical condition of the blood itself. We have seen that venous murmur in the neck is common in such cases; that the sounds of the heart continue; that the spleen is often in a state of congestion; and that the phenomena of "putrescence" are rarely developed. In these cases patients frequently go through their fevers without requiring wine or stimulants, and we have never observed any consequent alteration of the heart's structure. Debility there may often be, as shown by the prolongation of the first sound, but beyond this there is no recognised change of the organ.

RECAPITULATION.

1. That the heart, in common with other organs, is liable to suffer from organic and functional alteration in typhus fever.

2. That during the course of a particular epidemic we find cases otherwise similar, yet in which the state of the heart is different.

3. That cases of fever, considered with reference to the nervous condition of the heart, may be arranged into three groups:

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also Dr. Graves's Essays on the Use of Tartar Emetic and Opium in Fever. (Dublin Journal of Medical Science, First Series, vol. ix.)

in one there is excitement of the heart; in another, depression; while in a third there is no abnormal state of the organ beyond the variations of rate attendant on the different stages of fever.

4. That neither in its excited nor its depressed condition can the state of the heart be considered as inflammatory.

5. That a progressive loss of impulse, and also of the systolic sound, is the principal indication of the depressed state of the heart.

6. Conversely, a strong and jerking impulse, with distinctness of both sounds, indicates the excited condition.

7. That in neither of these conditions does the force of the pulse necessarily correspond with that of the heart. This is particularly seen in cases of the second group, in which a permanently excited heart may co-exist with a feeble pulse and cold extremities.

8. That in the cases of depression the disease is generally found to consist in a softened state of the ventricles, especially of the left.

9. That the opinion of Louis, as to the non-inflammatory character of this softening, may be adopted.

10. That this softening of the heart is commonly met with in cases where no analogous state of the voluntary muscles can be observed.

11. That it cannot be attributed to putrefaction, but may be held to be one of the secondary diseases of typhus.

12. That a weakened state of the heart, without softening or infiltration, may be admitted to exist. Such a condition is probably more frequent in the non-petechial than in the petechial fevers.

13. That the process of softening is not preceded by signs of excitement, nor has any case of re-active inflammation been observed in the maculated typhus of this country.

14. That neither in the advance nor retrocession of the disease is a correspondence between the impulse and sounds constant. The first sound may be greatly diminished, while the impulse remains; and during recovery, impulse may appear before sound, or sound before impulse.

15. That it is only by examining the patient when he is turned on the left side, and at the termination of the expiratory effort, that we can be certain of the complete loss of impulse.

16. That the value of loss of impulse depends on its progressive character.

17. That when the typhoid softening of the ventricle occurs in a heart already affected by mitral disease, and whose contractions are attended by mitral murmur, the latter may be suspended, but return during convalescence.

18. That the left ventricle, as is proved by the dissections of Louis and those conducted in the Meath Hospital, is the portion of the heart most liable to the disease.

19. And that the acoustic signs proper to the invasion and retrocession of the disease confirm this doctrine.[1]

20. That diminution, and ultimately cessation, of the systolic sounds at both sides of the heart may occur. The heart then acts with a single sound, which is the second.

21. That in a few cases the heart may continue to act without any perceptible sound.

22. That in such cases an extreme degree of the typhous softening has been found on dissection.

23. That in some cases the sounds of the heart in the adult resemble those produced in the fœtus in utero.

24. But that this condition, though implying great debility, is not so certain an indication of softening as that in which the first sound is especially diminished, or, it may be, lost.

25. That in some cases of softening of the heart a sighing respiration, and in others a tendency to syncope, may be observed.

26. That during recovery the signs of restoration appear first at the right, and next, at the left side of the heart.

27. That the second sound may be depressed, the cases being divisible into two classes: in the first both sounds are equally diminished, though neither are obliterated; while in the second the preponderance of the first over the second sound may be observed.

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[1] It will be remembered, that in a few cases we found on dissection not only that both ventricles were softened, but that the right appeared even more altered than the left. It is not improbable that future observations will establish a greater liability to softening of the right ventricle in the typhus fever of this country than in that of the continent. The great frequency of the secondary bronchial diseases in our cases should not be overlooked, and it may be inquired whether the same cause that affects the lung in typhus may not also influence the pulmonary heart. The observations of Rokitansky on the secondary bronchial disease in maculated typhus may be consulted.—*Op. Cit.* vol. iv.

28. That while extinction of the first sound, with preservation of the second, is common, we have met with no case of extinction of the second with preservation of the first sound.

29. That in a few cases of obliteration of the systolic impulse, an impulse at the base of the heart has attended the second sound.

30. That in certain cases in which the phenomena of softening had been marked, the pulse, during convalescence, has exhibited a remarkable diminution of its rate, falling as much as twenty or thirty beats below the natural standard, to which, however, in the course of a few days, it finally returned.

31. That in no case have we observed a rapidity of pulse during convalescence which could be connected with the previous softening of the heart.

32. That rapidity of pulse in convalescence generally indicates some latent visceral irritation.

33. That in certain cases of the worst maculated typhus an excited state of the heart may exist throughout the disease, although the pulse be feeble and the extremities cold.

34. That in other cases the excitement of the heart may be ephemeral, and intercurrent with the signs of debility, or may become developed after the progressive restoration of the first sound.

35. That this excitement does not proceed from any form of carditis.

36. That in a few cases loss or diminution of the second sound may be observed.

37. That the production of murmur, in connexion either with the excited or depressed state of the heart in maculated typhus, is of rare occurrence.

38. But that in a certain form of fever, characterized by the want of the symptoms of putrescence, the short duration of the fever, the absence of petechiæ, and the tendency to crisis and relapse, a systolic murmur, or, in some cases, a prolongation of the first sound, has been frequently observed.

39. That this murmur is a single murmur, and similar to that produced in ordinary mitral disease: it has not been observed with the second sound.

40. That it more frequently occurs in the relapse than in the

primary fever; it subsides gradually during convalescence, and its character and accompanying circumstances seem to point out that it is a form of anæmic murmur.

41. That in this condition the loss of systolic impulse and sound is rarely if ever observed.

42. That in the same class of cases we may observe, in place of a systolic murmur, a certain prolongation of the first sound, attended sometimes by a corresponding condition of the impulse.

43. That both the murmur and prolongation of the first sound become lessened in the upright position; but to this rule exceptions may occur.

44. That the change in the sound by the erect position was observed in a case of maculated typhus with musical systolic murmur. In this case the change was from a musical to a simple bellows murmur.

45. That the influence of the force of the heart upon these anæmic murmurs is still to be investigated. In one instance, the murmur was loudest when the heart was most weak; while in another, the reverse occurred. The first of these cases was one of maculated typhus, the second was an example of the short non-petechial fever.

46. That an excited state of the heart, attended by a rasping systolic murmur, not diminished in the erect position, has been observed in two cases of the short fever in patients who laboured under disease of the valves. In one there was an old mitral disease, in the other an acute endocarditis.

47. Lastly, that in the progressive diminution and ultimate loss of impulse, in the lessening or extinction of the systolic sound, or again, in the development of the fœtal character of the heart's sound in fever, we have direct and intelligible indications for the adoption of the stimulating treatment in fever.

APPENDIX TO THE PRECEDING CHAPTER.

When it is considered that this work is intended to be a treatise on that part of practical medicine which relates to diseases of the heart and lungs, rather than a dissertation on physical diagnosis, a brief statement of some opinions on the subject of fever, which appear justifiable, will not be useless in this place, inasmuch as they elucidate what has already been said on the condition of the heart in that disease.

The doctrine of essentiality has long been taught in the Meath Hospital; and with respect to the relation of the local diseases in fever to the general or parent affection, our opinions have been those entertained by most practical physicians. In believing in essentiality, we, after all, only follow the old masters in medicine, and though the frequency of the secondary diseases must be admitted, yet, on the other hand, we hold, that typhus fever may run its course, even to a fatal termination, without causing any known anatomical change.

With respect to the local diseases it has been always taught, that they were secondary to the general malady, so that the tumefied glands of the intestine, the swelling, softening, and purulent exudations of the bronchial membrane, the petechial eruption, and the softening of the heart, bear the same relation to the general malady, at least as to sequence and dependence, that the pustule in variola does to its parent disease.

If any change in my opinion has occurred of late, it is as to the question, whether the local diseases are themselves originally of an inflammatory character, or whether inflammation, when associated with them, is not accidental and re-active. To the latter doctrine, which has been so well illustrated by Rokitansky, we may assent; yet at the same time it is not proved that the local inflammations are in every instance re-active. And it is not unlikely that, in cases where symptoms of fever continue long after their usual period, the local diseases which spring up and multiply may be instances of inflammations unpreceded by any process of infiltration or softening.

The local diseases of fever, not only in every epidemic, but almost in every case of each epidemic, vary in their character, extent, seat, progress, and importance; and we may hold, that even when best marked, they are incompetent to explain the phenomena, not alone of maculated typhus, but of that apparently milder disease which, under the names of typhoid fever, relapsing fever, dothinenteritis, &c., has so long occupied the attention of physicians.

It is more than probable that the local affections which are not simply functional, and more especially those in which the process of retrocession has been interfered with by the occurrence of re-active inflammation, are an important cause of prevention of the critical, or at least, spontaneous, termination of the disease. The cessation of fever, after the application of leeches to the abdomen, on which the followers of Broussais have so much relied, is probably an instance of crisis being permitted in consequence of the removal or modification of a re-active inflammation which was interfering with the laws of periodicity. Finally, many circumstances, observable in the epidemic fevers of Ireland, must make us cautious in drawing too strong a distinction between not only the maculated and non-maculated cases of fever, but also between typhus, properly so called, and the typhoid disease already specified. It is not to be denied that the characteristic cases of either of these groups of diseases are different; and we have seen that with respect to the condition of the heart, which is the subject more immediately in hand, some remarkable contrasts have been found; yet we cannot help believing that these diseases are but varieties, and that they are results of the same poison or exciting cause acting on individuals in different states; but we need not here enter into lengthened discussions on this point. Let it suffice to indicate generally the grounds of this opinion; they are as follow:—

1. That epidemics of fever may occur in which, with well-marked petechiæ, the follicular ulcerations of the intestine exist in a large proportion of cases.

2. That in cases of the best-marked maculated typhus we have found occasionally the above anatomical condition, and this at a time when no epidemic disposition to the disease could be supposed to exist.

3. That we have frequently observed in cases where a large number of a single family have been successively attacked by contagious fever, that every form of the disease, from the most malignant typhus to the mildest typhoid fever, may be presented by different members of the family.

Lastly, that a patient who has gone through the typhoid fever, in its best-marked character, may, after an apyrexial period of a few days, be attacked with the most severe form of maculated fever, and conversely, that in certain cases, where the first fever was a maculated typhus, the symptoms in the second attack or relapse were those of the "typhoid fever."

These circumstances, when fairly considered, make a strong case against the existence in these countries of two essentially different forms of fever; and if anything was wanting to strengthen this conclusion, it is the great fact, that although in the so-called typhoid fevers we are not so often nor so urgently required to employ stimulating treatment, the general principles of managoment, in both forms of the disease, are truly the same.

CHAPTER VIII.

DISPLACEMENT OF THE HEART.

Although displacement of the heart cannot be ranked among the diseases of that organ, yet the study of its causes and accompanying phenomena will be found important.

These displacements are divisible into two classes, viz., the transverse and the vertical. A greater amount of attention has been paid to the first of these forms than to the second; for the transverse dislocation, especially when the heart is forced to the right of the mesian line, is a more striking phenomenon than mere vertical displacement.

The transverse and vertical displacements of the heart are indicative of disease of organs external to the pericardium. Indeed, I do not know of any case forming an exception to this rule, unless that in which, from an accumulation of fluid in the sac, the apex of the heart is found to impinge against different portions of the chest, according to the position of the patient.

We may also observe that, in individuals who have recovered from copious pleuritic effusion, a certain degree of laxity of the mediastinum sometimes remains, even for a considerable length of time, so that the heart, although there is no disease of the pericardium, changes its position in the transverse line according as the person turns to the left or right side.

Let us enumerate the principal causes of these transverse and vertical displacements, and then inquire how far the mere circumstance of dislocation affects the motions or the organic state of the heart.

Of the various forms of displacement, the transverse is the most common, and it is of two kinds,—one, where the organ by lateral pressure is forced into a new situation; and the second, when, in consequence of the diminished volume of one lung, it is drawn towards the affected side, in association with the surrounding parts.

The first variety is produced by the *diseases of accumulation*, or, in other words, by those affections which produce pressure from without. Of these the following are examples:—

1. Accumulation of liquid in the cavity of one pleura.
2. Accumulation of air, with or without liquid effusion, in the same situation.
3. Accumulation of air in the lung, as in extreme cases of Laennec's emphysema, when the disease greatly predominates in one lung.
4. The growth of tumours within the thorax, which, when they exercise a lateral pressure, easily displace the heart. Cancerous and aneurismal tumours, but more especially the former, produce this result.
5. The existence of hernia through the diaphragm.

It is very probable, that in many of the ordinary acute diseases of the lungs a certain amount of pressure, consequent on inflammatory turgescence, is exercised on the heart; and that if the disease was confined wholly to one side, there would be some displacement.[1] And it is probable that the latter often occurs, though to so slight a degree as to escape notice. As acute bronchitis is seldom confined to one lung, it is easy to understand that in this disease lateral displacement of the heart should be rare; but its absence is not so easily explained in acute pneumonia affecting one lung. In pneumonia the amount of tumefaction is not very great; and hence it is that in the differential diagnosis between pleurisy with effusion and mere pneumonia or pleuro-pneumonia, we owe so much to the evidences of eccentric pressure. I have never verified the displacement of the heart in acute pneumonia; but as it has been shown by Professor Smith that in *plastic pneumonia* enlargement of the lung may take place to such an extent as to press down the diaphragm, there is every reason to believe that the same condition might cause lateral displacement of the heart.

The occurrence of eccentric displacement, from tumefaction of the lung in acute pneumonia, has been established by Professor Smith. In a communication made in 1840, to the Pathological

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[1] Treatise on the Diseases of the Lungs and Pleura.

Society of Dublin, he brought forward the lung of a man who died of acute pneumonia. The disease had a peculiar anatomical character, and might be fairly described as a croupy inflammation of the most minute tubes and air-cells. When the lung was incised and washed, vast numbers of spherical granules of coagulated lymph were obtained in a perfectly detached condition. There was no suppuration; but so great was the enlargement of the lung that the diaphragm and liver were pushed far down into the abdominal cavity. The disease occurred in the right lung. Had it been in the left, the heart would, doubtless, have been displaced to the right side.[1]

In liquid effusions into the pleura, change of situation of the heart is seen at an early period, especially when the effusion is the result of inflammation. In this respect we observe a difference between hydrothorax and acute pleurisy; for in the latter the dislocation of the heart is unquestionably a more constant sign, and developed at an earlier period. How far this is, to be explained by any softening of the tissues, the result of inflammation, I shall not now inquire; but the displacement of the heart seems to be one of the very first signs of effusion. It may exist even before the upper portions of the chest have become dull, and is a circumstance of constant occurrence, long before any yielding of the muscular portions of the thoracic walls has taken place.

On the effect of aeriform collections in the pleura in displacing the heart we have as yet but little knowledge. But it appears probable that these can only act when, during expiration, the egress of the air is prevented. Of the signs of simple pneumothorax, without fistula in the lung, we know still less; yet we can safely say, that if a great accumulation of air were thus to occur, there could be no doubt that displacement of the heart would result.

In the ordinary case of air and liquid, it is probable that the dislocation is mainly owing to the pressure of the latter. If, however, the opening be valvular and not permanently patent, then the pressure of the liquid from below will, as it gradually increases, act on the superincumbent air, and thus the latter become an indirect cause of displacement.

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[1] See Dublin Journal, vol. xix. p. 122.

In Laennec's emphysema, the distending effect produced by acute bronchitis upon the already diseased lung is to be noted. An increase of volume, owing to the accumulation of air in the cells, may then be observed, and in certain cases, when the disease greatly predominates in one lung, transverse displacement is produced, which disappears on the subsidence of the attack. This, however, is not common, because the bronchial affection is seldom confined to one lung; and hence in Laennec's emphysema we more frequently observe the vertical than the transverse displacements. I have but little to offer on the subject of displacement resulting from the pressure of tumours, of which various examples may be found in pathological works. It is obvious that both the transverse and vertical displacements may be thus produced. In Dr. Houston's case cancerous tumours engaged both lungs, but particularly the right, and the mediastinum and heart were pushed to the left side.[1]

A remarkable example of vertical pressure is given by Boerhaave, in which an enormous tumour within the thorax so compressed the diaphragm that the heart existed under the umbilicus. This case is quoted by Testa, as also another from Meckel, in the Memoirs of the Royal Academy of Berlin, of a vast abdominal tumour which forced the heart to the right side of the sternum. Dr. Graves and I have published a case of aneurism of the abdominal aorta which presented in the left hypochondrium, and caused displacement of the heart to the right side.[2]

The last case of transverse displacement which I shall notice is that in connexion with congenital hernia of the abdominal viscera through the diaphragm. This condition has been most frequently observed in new-born infants who have lived but a short time after birth; but its existence is not incompatible with a considerable duration of life, as in a case by Weyland, quoted by Bouillaud, where the child lived for seven years, though liable to continual vomitings from the first period of its existence. The left side of the chest, as high as the second rib, was filled by the convolu-

[1] See the Catalogue of the Museum of the Royal College of Surgeons in Ireland.

[2] See Dublin Hospital Reports, vol. v.

tions of the intestines, and the lung was only one-sixth of its ordinary volume. The heart was situated in the mesian line.

A remarkable case occurred in Sir Patrick Dunn's Hospital, under the care of Dr. Osborne, which has been quoted by Dr. Graves and myself.[1]

A man about forty years of age died of tubercular phthisis.

The œsophagus, after passing through the usual opening in the diaphragm, was found to re-enter the thorax by another very large opening in the tendinous expansion towards the left side. The stomach, of which the cardiac and pyloric extremities were approximated, occupied the inferior portion of the left thoracic cavity.

A considerable portion of the transverse arch of the colon was also included in the left side of the chest; these viscera, loosely but permanently fixed by means of the serous membranes, all rested on the convex surface of the diaphragm, and had pushed the heart and mediastinum towards the right side. The margin of the unnatural opening in the diaphragm was formed by a round tendinous cord about the thickness of a quill, which added greatly to its strength, and was evidently of very ancient formation. The lung, small and tuberculated, did not exhibit any signs of compression, and was not adherent to the abdominal viscera. It may be easily conceived that the left pleural cavity was continuous with the cavity of the peritoneum, and both were lined by the one serous membrane.

This case is pregnant with interest: we observe in the adult *a new cause of displacement of the heart*, and a new source of difficulty in stethoscopic examination; for it is quite evident that auscultation applied to the left side of the thorax would have furnished very fallacious information, and the sounds heard would have varied according as the stomach and colon were full or empty. The same observation applies also to percussion; and the fact is, that during the life of this patient, those who examined his chest could not reconcile the phenomena afforded by auscultation or percussion with those of any known disease of the chest. The respiration was heard everywhere, except inferiorly and anteriorly on the left side, and here percussion gave a clearer sound than natural. No râle was audible in this part of the chest, but borborygmi and sounds resembling those produced by the motion of fluids in the intestines were observed.[1]

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[1] See Dublin Hospital Reports, vol. v.

In the first volume of "Medical Observations and Inquiries" a case is given of a child who died an hour and a half after birth. The spleen, as well as a large quantity of the intestines, was lodged in the left side of the thorax. Dr. Graves and I have observed that in most of the recorded cases of this condition death by obstructed respiration occurred within a few hours after birth; and Dr. Murphy has since given the particulars of another case, in which, although the heart continued to beat forcibly for some time after birth, there was not the slightest attempt at respiration, and every effort used to excite it failed. The child was above the average size, but had spina bifida, and both wrists were distorted. The whole of the left thoracic cavity was filled with the small intestines. The development of the left lung having been arrested

[1] "This man vomited frequently while under observation in the hospital. Now, as the stomach was placed entirely out of the reach of being compressed by the contractions of the diaphragm, and as this contraction completely defended it from the influence of the abdominal muscles, it is clear that in this case vomiting must have occurred indepeadently of the compression, either of the diaphragm or abdominal muscles. This fact, worth a thousand experiments, *completely decides the question, that vomiting may be produced by the action of the stomach itself, unassisted by any external compressing force,* notwithstanding what Le Gallois and late physiologists have said to the contrary." (*Op. Cit.*)

The most remarkable case of phrenic hernia, as regards the duration of life, is that recorded by Cruveilhier in the seventeenth Livraison of his Pathological Anatomy. A female, aged 75, who had been for many years subject to severe, but transient, attacks of colic, was admitted into the Salpêtrière under the following circumstances:—She was pulseless, and the surface was blue and icy cold. These symptoms were supposed by many to be those of cholera, but were referred by Cruveilhier to strangulation of an intestine, and the absence of any external tumour induced him to form the diagnosis of an internal strangulation. On examination, a large tumour, filling almost the entire of the left cavity of the chest, was found. The heart was pushed completely over to the right side. This tumour proved to be a large phrenic hernia, but the seat of the strangulation was proved to be in the abdomen, where the mesentery was seen twisted upon itself, and constricting a portion of the intestinal tube.

According to Cruveilhier, the deficiency of the diaphragm may exist from the time of birth, and yet no hernial protrusion occur for many years,—the liability to this event depending upon the position of the opening. If the latter corresponds to the situation of the liver, protrusion of any of the viscera may never occur; but if the deficiency in the diaphragm corresponds to any of the more moveable parts contained in the abdomen, the formation of a phrenic hernia is much more liable to take place.

almost at its commencement, the heart was larger than usual, and remained nearly in its natural situation. It is remarkable that in this case there was a deficiency in both alæ of the diaphragm, so that the right lung, which was of the ordinary size, was in close contact with the suprarenal capsule. The deficiency in development, however, was much greater on the left side.[1]

But whatever interest these cases, as well as those of congenital transposition of all the viscera, or of malposition of the heart only, may have in a physiological point of view, their rarity is so extreme that we need not discuss the subject at greater length. One important observation, however, may be made,—that in the case of imperfectly developed diaphragm,—in congenital dexiocardia, and again, in the transverse displacement from disease, the functions of the heart are not manifestly injured by the change of position.

DISPLACEMENT FROM DIMINISHED VOLUME OF THE LUNG.

In the preceding cases of displacement of the heart the change has been effected by pressure, as in empyema, dilatation of the air-cells, or a tumour. How far pressure acts in the case of hernia through the diaphragm is still to be determined. And in the congenital malposition, with or without transposition of other viscera, we cannot attribute any effect to pressure.

But there is another and equally important displacement which arises from opposite mechanical conditions. This is observed in cases in which, from any cause, the volume of one lung undergoes much diminution. As this change commonly affects but one lung, it may be that the heart is not only drawn towards the lung so engaged, but also forced in that direction by the increasing volume of the opposite organ. However this may be, it is certain that both the transverse and vertical displacements are thus produced. These cases may be called the concentric displacements of the heart, in contradistinction to the eccentric, or those where the heart is displaced in the diseases of accumulation. Between these opposite cases some differences are to be observed: the eccentric displacements may occur at an early period of acute disease, and their extent is a measure of the accumulation. They vary, with its amount, and disappear either wholly or in part, on the subsidence of the original disease. The concentric displacements, on the other hand, are generally the result of some chronic disease which produces atrophy of the lung. Of the mere atrophy, the amount of dislocation may be a measure, but it is not so of the degree or quantity of disease; and when the concentric displacement is once produced, the organ, so far as we know, never returns to its natural position.

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[1] Reports of the Obstetrical Society of Dublin, Dublin Journal of Medical Science, vol. xv. Cruveilhier observes that arrest of development of the diaphragm is much more frequent at the left than the right side.

The conditions in which we have observed concentric displacement of the heart are the following:—

1. Chronic tubercle of the lung.
2. Chronic gangrene of the lung.
3. Atrophy of the lung with dilatation of the bronchial tubes,—the cirrhosis of the lung of Dr. Corrigan.
4. Diminished volume of the right lung, consequent on the absorption of an empyema.

Of these the last is the most frequent; and by it are to be explained many of the examples of the heart pulsating at the right side, in persons who enjoy perfect health. These individuals, at some time long past, have suffered from an empyema of the right pleura, and, owing to the circumstances which we shall now indicate, the heart has passed across the mesian line, and become permanently fixed in its new position.

A remarkable case of displacement of the heart to the right side was published by me several years ago, as an example of this condition resulting from violence. At that time the form of concentric dexiocardia, now under consideration, was unknown to me. As full details of this case have been given, it is unnecessary to say more than that the heart was permanently fixed in the right mammary region, where it was observed soon after a terrible accident, by which the chest had been severely injured, and which had caused extensive pulmonary inflammation. I now entertain scarcely a doubt that a pleuritic inflammation of the right side occurred, and that by the rapid absorption of the fluid then effused,—the lung being unable from various causes to recover its original bulk, the heart passed over, and became fixed in the right side of the chest.[1]

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[1] See my original Paper in the Edinburgh Medical and Surgical Journal; and also my "Treatise on the Diseases of the Lungs and Pleura," p. 308.

The case in which, so far as I know, this condition was first observed is given in my work on Diseases of the Chest, and is remarkable as being an instance of dexiocardia, the result of acute disease of the right pleura. The displacement followed on the rapid absorption of an acute empyema.

The remaining cases of concentric displacement to the right side are examples of diminished volume of the lung from disease of its structure. We can understand that in chronic tubercle the transverse displacement will be rarely observed, as it so seldom happens that the disease is confined to one lung. I have observed it, however, in a few cases where the left lung had resisted diseased action for a considerable period. There is also another reason why this form of displacement should be less seen in tuberculosis of the lung than in other cases, which is, that the early formation of cavities, by permitting expansion of the diseased lung during inspiration, prevents the diminution of the volume of the organ. The displacement, then, is only in proportion to the amount of the atrophy of the pulmonary structure, and when occurring after the formation of cavities, it may be a measure of the extent of their cicatrization.

The vertical displacement upwards is more common from obvious reasons. I have seen a case of extremely chronic phthisis in which the diminution of volume of the left lung was so great that the heart pulsated in the infraclavicular region. In this instance the disease had lasted for more than five years. No post-mortem examination was made, but the atrophy of the lung must have been extreme.

I communicated to the Pathological Society in 1850 an example of transverse displacement to the right side in a case of chronic gangrene of the lung.

But the most common instance of this form of displacement is that disease to which Dr. Corrigan has given the name of cirrhosis of the lung, and which consists in a dilatation of the larger bronchial tubes, with obliteration, more or less perfect, of the intervening air-cells, and attended with diminution of the whole volume of the lung. I believe that the history of this condition is not yet fully established.

Reasoning from analogy, we may admit that a disease of the lung, similar to the cirrhosis of the liver, does really exist. This would give a cause for dexiocardia, or its opposite.

In many cases of absorption of a pleuritic effusion, where the lung does not regain its original volume, dilatation of the bronchial tubes takes place, and advances slowly till a condition, mechanically similar to cirrhosis of the lung, as described by Dr. Corrigan, actually occurs, and then we have a case which, when seen for the first time, is difficult or impossible to be distinguished from the original disease. The patient has symptoms of chronic bronchitis, rather than of phthisis, yet he is subject to hemorrhages from the lung. The upper portion of the right lung gives the signs as of anfractuous cavities, and the heart beats to the right of the sternum.

Finally we observe, that in all these examples of concentric dislocation of the heart, as compared with those from eccentric pressure, there is this difference, that the heart never returns to its natural position. In the eccentric cases the reverse occurs, as every one who has studied the absorption of an empyema must know. It is true, that in some chronic cases of the latter disease, the heart does not absolutely regain its original position, yet it retraces its steps to a great extent.

Considered with reference to the probable duration of life, this rule may be laid down,—that when in any case of supposed phthisis, in which the physical signs show disease of the upper portion of the right lung, we find the heart to the right of the sternum, and at a point of the chest higher than natural, we may predict a longer duration of life than in cases where this symptom does not exist; for the chances are greatly in favour of the existence of one of two conditions of the lung, either of which imply a slowly advancing disease. The first is the cirrhosis of Dr. Corrigan,—a condition under which, as is well known, patients may exist for years. The second may be described as follows:—An individual, previously healthy, is attacked with the symptoms of tubercular deposition in the superior portion of the right lung,—the disease goes on, and the physical signs pass through the usual changes, from those which indicate the earlier stages of tuberculous irritation to those of a cavity. So far there is nothing unusual to be observed. But in some instances, whether from the influence of treatment or the powers of nature, the usual course of disease is interrupted. The tendency to new tubercular deposition becomes indolent, or, it may be, ceases; the constitutional symptoms are less marked; and the patient may gain in flesh and appearance. The abdominal viscera, and, in many cases, the opposite lung, seem to escape disease, and thus a patient whose case was considered likely to terminate fatally within the first twelve months of his illness goes on in a doubtful state of health, for two, three, or more years. After the second year it may happen that, although the upper portion of the lung retains its dulness on percussion, and exhibits a very deficient vesicular murmur, generally combined with mucous râles, yet the signs of cavity wholly, or in a great measure, disappear, and the heart is found pulsating to the right of the sternum. This displacement is probably owing, on the one hand, to atrophy of the lung, and on the other, to the more or less complete cicatrization of the cavity. As has been already remarked, such a case, seen for the first time, would be with difficulty distinguished from true cirrhosis.

But putting this aside, the existence of these two forms of disease, and their chronic nature, justify the practical conclusion, that in any case exhibiting physical signs of disorganization of the upper part of the right lung, we should, if the heart be found at the right of the sternum, expect a longer duration of life than in others where this displacement has not occurred.

We may divide the examples of concentric dexiocardia, from all causes, into two classes. In the one, the patient continues to suffer from the effects of the pulmonary disease which has induced the displacement, and the true symptoms of chronic disease of the lung are established. In the other, although the heart continues in its new position, the individual remains altogether free from pulmonary disease.

In the first category of cases we may enumerate the following:—

1. Displacement consequent on atrophy of the lung, and dilatation of the large tubes.
2. Displacement, following on tubercular disease.
3. Displacement, the result of empyema of the right side, the absorption of which is not followed by the restoration of the

volume of the lung, and which organ passes into a state which, if not the same as the true cirrhosis, is closely allied to it.

In the cases of the second category it appears that, after the absorption of a pleuritic effusion and the displacement of the heart, the lung undergoes no farther change. It is diminished in volume, but that is all; and in such instances, the most perfect health may exist for the ordinary periods of life, although the heart be always found to the right of the sternum. I have observed several of these cases, and in them all the original attack of pleurisy occurred at an early age, although in some of them the affection had been at the time overlooked. Finally, it may be stated, that we have found no instance of organic disease of the heart which could be traced to the circumstance of its displacement.

It has already been noticed, that in the eccentric displacement of the heart in ordinary empyema, the organ suffers but little from the effect of pressure. This could hardly have been anticipated. In some chronic cases of empyema it is most singular to observe the tranquillity of the heart's action, even when it is pulsating far to the right of the mesian line.

I have published a case of eccentric dexiocardia in which an attack of pericarditis supervened before death, and yet the heart's action was in no way disturbed. This is a most singular fact. In the concentric cases we cannot suppose that there is any important pressure exercised on the heart, and its freedom from functional disturbance is more easily understood.

A remarkable case of vertical as well as lateral pressure on the heart has been recorded by Dr. Adams.

A man, aged 26, was admitted into the Whitworth Hospital with symptoms of chronic bronchitis, attended with signs of empyema. Active measures failed in relieving him, and the fluid in the chest appeared to increase. After some time he was exposed to cold, and all his symptoms became aggravated. The dyspnœa increased; the face became livid; the jugular veins were distended, and he was unable to expectorate. Erysipelas of the right arm set in, and a large gangrenous spot subsequently made its appearance. On the nineteenth day after admission Dr. Adams found him sitting up in bed, holding by the sides of his bedstead, and gasping for breath. His eye-balls were protruded, and his face livid, and bathed in cold perspiration.' A reddish serum was flowing from his mouth, and respiration was inaudible, except at the upper portion of the right lung. The *belly was swollen and tympanitic.* In this state he remained for eighteen hours, and died on the following night.

On dissection, both pleuræ contained a great quantity of bloody serum. The left pleura was completely filled by it, and the lung, compressed and carnified, adhered to the posterior portion of the chest. The respiratory process seemed to have been carried on almost wholly by the upper third of the right lung. The heart lay compressed, and in a horizontal direction. The right ventricle was folded on itself, and the entire organ was flattened by pressure. The right ventricle and the pulmonary artery were empty, and the ventricle itself was thrown into three rounded folds; and in the line of these folds the parts of the heart which were in apposition were firm, and had an appearance as if parboiled, showing that the folding of the ventricle was a condition of some standing.[1]

In this case the heart was exposed to an extraordinary pressure, for not only were both pleuræ distended by liquid, but the descent of the diaphragm was interfered with by the abdominal swelling. In this respect the case differs remarkably from those already alluded to, in which, notwithstanding great displacement of the heart, the organ did not appear to suffer any disturbance, mechanical or vital. It will be recollected, that in these instances the effusion was confined to one pleura.

The horizontal direction of the heart should be noted. Dr. Corrigan has described this result in cases of hypertrophy and dilatation with a permanently patent aortic opening;[2] and this case gives us an additional cause for the change in question.

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[1] Transactions of the Pathological Society of Dublin, May 30, 1846. Dublin Journal of Medical Science, First Series, vol. xix. page 822.

[2] On Permanent Patency of the Aortic Valves. (*Op. Cit.*)

CHAPTER IX.

RUPTURE OF THE HEART.

If we exclude the ruptures of the heart from external violence, of which various instances are on record, we may enumerate the following examples of the accident:—

1. Rupture of the ventricles or auricles, with effusion of blood into the pericardium.
2. Rupture of the inter-ventricular septum.
3. Rupture of the valves, or of the tendinous cords.

The general principles of diagnosis of internal solutions of continuity are applicable in these cases; for we observe the sudden development of new, extraordinary, and often fatal symptoms to attend most instances of the accident.

As might be expected, rupture of the muscular portions of the heart, not preceded by organic change in the part which gives way, is of extreme rarity. This is at least true with respect to the walls of the heart. Muscular fibre is, for obvious reasons, endowed with an extraordinary power of resisting rupture. Were it otherwise, the accident would be common in many cases, especially of the hysteric excitement of the heart. In tetanus, it is true that rupture of the voluntary muscles has been observed; but it is probable that the heart, when under excitement, is, from the very nature of its action, less liable to be torn than the voluntary muscles.[1] A case is recorded by Harvey of laceration of the heart, apparently from over-action. The patient had disease of the aortic orifice and hypertrophy of the left ventricle. But this case is to be considered as one of rare occurrence, and it is probable that the hypertrophy was succeeded by a softening process, such as would occur in an early stage of the fatty degeneration.

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[1] Laceration of the abdominal muscles is not unfrequent in tetanus. An example is recorded by Dr. Latham in his work on "Diseases of the Heart." The accident has been often observed in the Dublin Hospitals. It may occur in other cases of violent spasm of the abdominal muscles. Thus, it has been met with by Professor Smith in cases of retention of urine from stricture of the urethra. In some of such cases the recti muscles were actually torn across. The same observer has found the heart in tetanus not only so firmly contracted as to cause obliteration of the cavity of the left ventricle, but twisted on itself so as to present a spiral condition from the extreme contraction of the oblique muscular fibres. The action of tetanus on the involuntary muscles is still to be investigated.

The following conditions predisposing to rupture of the heart may be enumerated:—

1. Abscess in the walls of the heart.
2. Apoplectic effusions into the substance of the heart, as described by Cruveilhier.
3. Muscular aneurism.
4. Fatty degeneration.

Of these it is hardly necessary to observe, that the last is the most frequent. But a common character belongs to them; for in all there has been a pre-existing chronic disease of the heart; and, from the solution of continuity, a fatal effusion of blood into the pericardial sac. It is, indeed, a matter of wonder, when we consider the frequency of fatty degeneration of the ventricles, that the accident is not more frequent, and its rarity can only be explained by referring to the diminished force of the muscular contraction.

Rupture of the walls of the heart may occur in either ventricle, or in the auricles. On the relative frequency of the accident in the right and left ventricle it is unnecessary, so far as practical medicine is concerned, to enter. Let it suffice to say that, in some cases, several lacerations have been found; and again, that the effusion of blood is not always so copious as to cause sudden death by syncope, but that it may be delayed or arrested, just as in cases of aneurism, by the formation of a coagulum, which in part, or altogether, blocks up the rent in the cavity which has given way. The lesion, as might be expected, is most frequently met with in old persons, and may be diagnosticated under the following circumstances.

A patient in advanced age has laboured under some of the symptoms and signs of organic disease of the heart. Evidences of aortic aneurism are absent, and the disease seems to be some form of a weakened heart, with or without the characteristic signs of fatty degeneration. Suddenly, perhaps after exertion, a new train of symptoms occurs; the dyspnœa increases; anguish and syncope set in; the pulse fails; the præcordial region becomes more extensively dull, and death supervenes. But even all these circumstances would only justify a strong suspicion of the occurrence; for in cases of weakened heart, where no rupture has taken place, the symptoms attendant on the fatal termination are often very similar to those observed in cases of rupture with effusion of blood into the pericardium.

We owe to Dr. Bigger, of Dublin, one of the most instructive cases of rupture of the left ventricle on record. It was communicated by that gentleman to the Pathological Society of Dublin in 1841. The patient, a man aged 31, had resided in the West Indies for a considerable period of time. In 1835 he exhibited symptoms of incipient phthisis, but there was this remarkable circumstance, *that the pulse would frequently fall from the rate of fifty to that of fifteen in the minute, and so remain for eight or ten hours.* In the course of two years the patient was again seen by Dr. Bigger, who found him labouring under symptoms of advanced phthisis, and with the signs of cavities in both lungs. He was subject to fits of despondency, and it was during these that the retardation of the pulse used to take place. *When the pulse was thus slow, a soft bellows murmur attended the first sound, but ceased on the heart's action being restored to its natural standard.* It is to be remarked, that this symptom existed from an early period of the affection. On the 2nd of March, 1841, the general symptoms having been for some time getting worse, Dr. Bigger was suddenly called to see him. He found him in extreme distress, kneeling by the bed-side, and gasping for breath. Death occurred in a few hours.

On dissection, both lungs were found extensively tuberculated, with cavities existing at their summits. The pericardium was full of fluid blood, and in the left ventricle there was a ragged aperture, through which a portion of a fibrinous clot protruded. This appeared to have been forced through at the first rush of blood, and was part of a long coagulum which had partly passed through the opening. Around the orifice the muscular fibre was discoloured and broken down; large coagula, some of which exhibited a distinct hollow in the centre, were entangled in the carneæ columnæ.[1]

In this case we have a good example of the combination of chronic tubercle of the lung with, in all probability, a fatty degeneration of the heart, and the symptom, so unusual in ordinary phthisis, of the diminution in the rate of the pulse, occurring from time to time, is worthy of being noted.

It will be remembered that, notwithstanding the laceration of the ventricle, the death was not sudden. We have, perhaps, been too much disposed to consider the circumstances of the rupture of the heart, and the consequent effusion of blood into the pericardium, as analogous to those of the rupture, in most instances, of aneurism, and hence to wonder why it happens that death is not always sudden in such cases. But when we consider that in the case of the heart the cavity in which the rupture occurs is that of a muscular organ, alternately contracting and relaxing, so that the escape of blood is, in all probability, intermittent; and again, that the pericardium is less capacious than either the pleural or peritoneal sac, we may understand why death should not always occur suddenly in rupture or wounds of the heart.

Further, when a certain quantity of blood has escaped into the pericardium, this cavity in its mechanical relations, may be held to represent an aneurismal sac; and the pressure of the fluid, especially if the opening be at all valvular, will tend to close the orifice, and in this way promote the formation of a clot. In the pleura or peritoneum, from the great capacity of these sacs, such a quantity of blood is commonly effused, as, in most cases, to produce immediate death.

It is hence difficult to offer a perfectly satisfactory explanation of the occurrence of sudden death in cases of rupture of the heart. The result in question must be referred to several causes, of which the following are the most evident:—

1. The sudden withdrawal of a certain quantity of blood from the system at large.

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[1] Transactions of the Pathological Society of Dublin, 1841. Dublin Journal of Medical Science, First Series, vol. xxi. page 300.

2. The effect of this loss on a heart already debilitated by the disease which has predisposed it to rupture.

On these points, however, the views of Cruveilhier must be noticed. This pathologist is of opinion, that death in cases of rupture of the heart is not to be ascribed to the amount of the hemorrhage; for the inextensible pericardium, which forms a shut sac, confines the effusion within narrow limits, and the patient dies neither of the hemorrhage nor of the sudden disturbance in the functions of the heart caused by the perforation, but really in consequence of the arrest of the circulation produced by the compression of the organ.

When, however, we reflect on the amount of pressure which the heart sustains with impunity in other diseases, we cannot altogether subscribe to the opinion, that in rupture of this organ, death is to be attributed solely to the pressure of the effused blood. On the other hand, it is to be remembered that the pressure is sudden; and again, that its influence must, of course, be greater than where the heart is neither ruptured nor debilitated by previous disease.

In the great proportion of cases the left ventricle has been the seat of the lesion, and the perforation has occurred either at or near to the apex. It is here that, not only in the normal state of the heart, but in hypertrophy, the left ventricle is found thinnest. Cruveilhier, indeed, inclines to the opinion that rupture occurs exclusively in the left ventricle; but from a table drawn up by Dr. Townsend it appears that in twenty-five cases of rupture of the heart, there were examples of rupture of the right ventricle; and of nineteen cases collected by Bayle, there were also three in which the lesion occurred in the right ventricle.[1]

Thus, out of forty-four cases, in six the rupture occurred in the right ventricle.

An additional example has been given by Professor Smith. The patient had for two years previous to his death suffered from repeated attacks of rheumatism, but had never complained of any affection of the heart until the night of the 11th of November, 1839, when he was suddenly seized with symptoms of collapse.

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[1] See Dr. Townsend's Memoir, Dublin Journal of Medical Science, vol. i.

and præcordial anxiety. His pulse fell to 40 in the minute; his extremities became cold; the countenance was pale; and the whole surface bedewed with a cold perspiration. In this state he remained for nineteen hours, and died on the next day.

The pericardium was found distended with blood, and a small lacerated opening was seen in the apex of the right ventricle, near to the septum. The parietes of the ventricle became gradually thinner towards the seat of rupture. There was no other disease.

The predisposing cause of this accident may be considered to be atrophy of the heart, with or without the fatty degeneration. Perforation of the walls of the heart, by the bursting of an abscess, cannot be strictly looked upon as a case of rupture; and with respect to the apoplectic effusions mentioned by Cruveilhier, I am inclined to consider them rather as the consequence of a previous laceration of the muscular parietes on their internal surface, than as themselves the cause of the solution of continuity.[1]

SOLUTIONS OF CONTINUITY OF THE INTERNAL PORTIONS OF THE HEART.

Rupture of the tendinous cords of the auriculo-ventricular valves.—This accident more frequently occurs to the mitral than to the tricuspid valves. When the greater frequency of organic change in the valves of the arterial side of the heart is borne in mind, we can understand why rupture of the tendinous cords should occur more frequently at the left side. For, as it must be admitted that in spontaneous rupture of the ventricle some diseased action has preceded the laceration, so in that of the valves, or tendinous cords, we may believe that a process, tending to increase their friability, predisposes them to rupture on being exposed to tensions which, had the parts been healthy, would have been safely borne.

In a practical point of view, we may divide the conditions predisposing to rupture into two classes,—one, the softening and atrophy of the valves; and the other their induration from fibrous or atheromatous degeneration. Of these, the latter is by far the most frequent; and the change, which in many cases arises from a chronic endocarditis, affects the valves, the tendinous cords, and the endocardium investing the musculi papillares. When rupture takes place, the valves themselves may be lacerated, or the injury be confined to one or more of the tendinous cords. It is probable that the latter case is the more frequent.

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[1] "Anatomie Pathologique du Corps Humain, par J. Cruveilhier," Livraisons xxii. xxx. See also Dr. Smith's Contributions to the Pathological Anatomy of the Heart and Great Vessels, Dublin Journal of Medical Science, vol. ix.; and Dr. Quain's Memoir on Fatty Degeneration of the Heart, Medico-Chirurgical Transactions, vol. xxxiii. p. 140. Dr. Latham's work on Diseases of the Heart, Lecture xxvi., should be consulted.

The occurrence of this accident, like that of other internal solutions of continuity, is often suddenly revealed by formidable symptoms; but, as in perforations of the hollow viscera, it sometimes—and probably with greater frequency than has been supposed—is unattended with any remarkable aggravation of the former symptoms, or production of new phenomena. We cannot explain these circumstances any more than when they are observed in perforation of the pleura or peritoneum; nor tell why in one case of rupture of the valves, or the chordæ tendineæ, there is disturbance of the heart's action, dreadful anguish, and speedy death; while in another we find these ruptures, without there having been anything in the symptoms to indicate at what period the accident took place.

As might be expected from anatomical relations, and also from the greater liability to inflammation and organic changes of the left, as compared with the right side of the heart, rupture traceable to the effect of force is much more common in the auriculo-ventricular valves than in those of the aorta or pulmonary artery. The accident may be practically considered as engaging the mitral valves and their tendinous cords. It is probable that in many cases of extreme alteration of the mitral opening, successive ruptures of the tendinous cords have occurred during the process of thickening and shrivelling of the valves. However this may be, the cases of rupture of the mitral valves are divisible into those attended with sudden and extreme distress, and those in which it is difficult or impossible to ascertain the precise period of the accident.

The following case is a good illustration of the first of these forms:—

CASE LXVII.—*Long-existing Hypertrophy of the Heart, with mitral murmur; Acute Pleuro-pneumonia, attended with extreme excitement of the Heart; Sudden occurrence of symptoms of cardiac anguish; Death; Laceration of the mitral valves and their tendinous cords.*

A lad, aged 15 years, had long laboured under symptoms of hypertrophy of the heart. The disease seemed to have commenced by an attack of pericarditis several years before. He was admitted into the Meath Hospital with all the indications of acute pneumonia in a high degree. The left lung was extensively engaged, and its lower portion had passed into hepatization. In addition to the symptoms and physical signs of the most sthenic form of pneumonia, we found that the action of the heart was violent and extended, while an immense bellows murmur was heard with the systolic sound, at the left side of the heart. It was plain that we had to contend with symptoms of violent pneumonia in a case of great hypertrophy of the heart. Active depletion was used, and the patient was placed under the influence of tartar emetic. The violence of the symptoms was greatly reduced, and auscultation showed commencing resolution of the disease of the lung, when, as is believed, from some indiscretion in regimen, the symptoms of pneumonia returned with, if possible, increased violence. On the second day of the relapse the heart was acting with great force, when suddenly the patient sprang up in bed, screamed as in agony, and exclaimed that his "*heart was broken.*" He pointed to his heart as the seat of his distress, and after suffering a few hours from terrible cardiac anguish and orthopnœa he expired.

The chordæ tendineæ were almost all ruptured near to their insertions into the mitral valves. The valves themselves were indurated and thickened, and one of them was lacerated through its whole extent. The left ventricle was hypertrophied and dilated, its muscular structure being red and firm.

We owe to Corvisart the first notice of rupture of the tendinous cords of the mitral valve, and there is some similarity between the first of his cases and that now given. In both the ruptures took place during an access of pneumonia; but in the case by Corvisart the heart was not hypertrophied; and it was difficult to determine at what exact period of the disease the laceration took place. The patient, after having undergone great fatigues, was attacked with symptoms of pneumonia, and had been largely bled before he entered the Hospital of La Charité. This was on the eighth day of his illness. The pulse was then small, hard, frequent, and irregular. The impulse of the heart was strong, but, in addition to these, to use the words of Corvisart, "On sentait un battement confus et irrégulier qui ne ressemblait en rien aux mouvemens du cœur."

The patient was in a state of extreme agitation and restlessness. The symptoms went on increasing, and during the next night he was terribly agitated, sitting down and rising unceasingly, and struggling with the impending suffocation. His sufferings continued up to the period of death.

Before the body was opened, Corvisart made the diagnosis of an acute lesion of the heart, and of rupture or laceration of some of its structures.

The upper portion of the right lung was hepatized, and there was interlobular pleurisy. The heart had not acquired any extraordinary volume, and it was found that one of the large fleshy columns in connexion with the mitral valve was torn across at its base, so that it floated freely in the cavity of the ventricle at the portion of the wall of the heart from which it had been torn.[1]

The following case was communicated by Dr. Gordon to the Pathological Society, and I am indebted to that gentleman for permission to insert it in this work.

CASE LXVIII.—*Permanent patency of the aortic valves, with hypertrophy and dilatation of the left ventricle; Rupture of the chordæ tendineæ of the anterior portion of the mitral valve.*

A man, aged 27, had eleven years before suffered from an attack of rheumatic fever, from which, however, he recovered, and was able to engage in the occupation of a field labourer up to the summer of 1850. On the 17th of June, after having been repeatedly over-heated and being probably exhausted by carrying a heavy weight, he was a second time attacked with articular rheumatism. This illness was but of short duration, and during its continuance there was no suspicion of any disease of the heart. Five months having elapsed, he came under the care of Dr. Faussett, complaining of a sensation of "weight in his stomach," and of inability of exertion. Dr. Faussett detected the usual signs of a permanently open aortic orifice, with hypertrophy and dilatation of the left ventricle. Towards the end of the month, in an attempt to run for shelter from heavy rain, the palpitation of his heart became unusually violent, attended with a choking sensation in his throat, which lasted for several hours. Up to this period the disease had been making slow but steady progress, but thenceforward its character was changed. He had been perfectly free from pain, but he now suffered from a stitch in the left side, and a distressing sensation of tightness across the chest. The palpitations, which used only to be excited by exercise, became incessant, and were attended with nausea, frequent vomitings, and cephalalgia. He had frequent cough, with red, frothy expectoration, permanent difficulty of breathing, and a constant feeling of impending suffocation; but his greatest suffering was from want of sleep, caused by an inability to lie down, or even to remain for any length of time in one position.

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[1] Two more cases are given by Corvisart in which the exact period of the accident was not accurately determined. In one the patient lived for twenty months after the presumed period of the rupture. The exciting cause seemed to have been an over-exertion in lifting a heavy weight. Two of the chordæ tendineæ of the mitral valves were broken. The points at which the rupture took place were smooth and rounded. In his third case (Obs. 33) the question of rupture by actual violence is doubtful. ("Essai sur les Maladies et les Lésions Organiques du Cœur," &c. By J. N. Corvisart, pp. 226, 227.)

He was admitted into the Whitworth Hospital on the 24th of December, 1850. He was then pale and anæmic, with a countenance expressive of pain and anxiety. His voice was very feeble, and he could only speak in whispers. His restlessness, and the other above-mentioned symptoms, continued unabated. The pulse was intermitting, small, and frequent; the extremities cold, and covered with a clammy perspiration. He had also anasarca and ascites.

The præcordial region sounded dull from the second to the seventh rib, and transversely from the junction of the cartilages of the ribs on the right side to a point two inches beyond the left nipple. There was also comparative dulness at the base of the right lung. The remainder of the chest sounded well. The respiratory murmur was feeble, with scattered bronchitic râles, and a large crepitus existed in the postero-inferior portion of the right lung.

In addition to the signs of defective aortic orifice and enlargement of the left ventricle, Dr. Gordon observed the following phenomena:—

An intense fremitus existed over the heart, having its point of greatest intensity at about the centre of the præcordial region. A bellows murmur, which, though variable in intensity, could not be localized, was heard all over the chest. It was, however, easily distinguished from the double murmur proceeding from the aortic valve, and was nearly as loud in the posterior as in the anterior portions of the chest. He died on the 26th of November, during a violent paroxysm of orthopnœa.

The pericardium was found to occupy a space extending from the sterno-clavicular articulation to the seventh rib, and transversely from the junction of the ribs with the cartilages on the right side, to an inch beyond the left mamma. The serous membrane was healthy, and the sac contained about three ounces of serum. The apex of the heart appeared to be entirely formed by the right ventricle, which, with a small part of the right auricle, were the only portions of the heart seen on opening the chest. The right auricle was enlarged, and the tricuspid opening was nearly twice its natural width; the valves were healthy. The right ventricle was very much dilated, and its walls thickened; the orifice of the pulmonary artery was slightly enlarged, but the valves were healthy.

The left auricle was found of nearly twice its natural dimensions; its walls were thickened. The left ventricle was enormously dilated, with great thickness of its parietes; and the mitral orifice so enlarged as to admit four fingers as far as the second joint; the cords of the anterior portions of the valve were all broken across near to the fleshy columns; they were thickened, softened, and covered with beads of very soft lymph. The aortic orifice was dilated, and the valves thickened and cartilaginous, particularly the posterior valve, which was more vascular, and covered with beads of lymph. The endocardium, between the aortic and mitral valves, presented the same appearance, but was otherwise healthy, as was the aorta. The bronchial tubes were thickened and vascular, and filled with mucus, and the posterior portion of the right lung congested. The spleen was enlarged, and the peritoneum full of fluid.

In reviewing the circumstances of this case, so accurately observed by Dr. Faussett and Dr. Gordon, we cannot help coming to the conclusion, that the rupture of the tendinous cords of the mitral valve was of recent occurrence, and produced by the tumultuous action of the heart excited during the patient's attempt to run home. For then a new class of symptoms was developed, which continued without intermission till the period of death. It will be remembered, that the ruptured cords presented beads of soft lymph, and that the endocardium, in the vicinity of the valve, was vascular. The pulse, too, when the patient came under Dr. Gordon's care, was small, feeble, and intermitting; and these circumstances, taken in connexion, on the one hand, with the previous history of the case, and on the other, with the extraordinary thrill and murmur heard all over the chest,—a murmur evidently different from that which was seated over the aortic orifice, led Dr. Gordon to diagnosticate that, in addition to the usual results of a permanently patent aortic valve, there would be found a great enlargement of both auriculo-ventricular orifices. The correctness of this opinion was established by dissection; and now, with this case on record, it will be for future observers to determine whether, in a case of a well-marked aortic regurgitation, the sudden occurrence of new symptoms, or extreme aggravation of those already existing, attended by signs of pulmonary congestion, a change in the character of the pulse, with a development of a new and extended valvular murmur, may not be taken as diagnostic of rupture of the cords or valves in one of the auriculo-ventricular openings.[1]

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[1] A remarkable prominence of the anterior portion of the left side was observed by Dr. Faussett on the 15th of November, and at that time there was a thrill communicated to the hand. This prominence afterwards subsided. Nothing afforded the patient relief but venesection, which was practised on three occasions, the blood on the first two being inflammatory. It is more than probable, that at that time the thrill proceeded only from the disease of the aortic valves, as it may arise under these circumstances either during the egress or regurgitation of the blood.

RUPTURE OF THE TRICUSPID VALVES.

Of this accident I have never seen an instance; and all writers agree that it is a circumstance of extreme rarity. And when we consider the physiological as well as the pathological relations of the right cavities of the heart, we can understand why rupture of the tricuspid should be so much less frequent than that of the mitral valves.

Professor Todd has enriched medical science by an important case of this accident.[1] In the remarks which precede his narrative Dr. Todd has the following observations:—

"My experience has long since led me to the opinion that it is an error to suppose that the right side of the heart has any peculiar exemption from disease, as Bichat affirmed, and his followers repeated. The truth of this matter seems to be, that in the chronic morbid changes to which the valves are liable, due to deposits of abnormal matters, whether interstitial or superficial, the right side is later than the left, and that it is not uncommon to find both sides similarly affected, the valves of the right side presenting an earlier stage of the deposit than those of the left; but that acute affections of the endocardium are of extremely rare occurrence in the right ventricle or auricle. The greater force exerted by the left ventricle of the heart, estimated by Valentin at one-fiftieth of the weight of the body, while that of the right ventricle is only one-hundredth part of the same, and the greater quantity of muscle in the former than in the latter, estimated by the same physiologist to be in the left ventricle double that of the right, denote a greater activity of nutrition on the one side than the other, and, therefore, a greater liability to abnormal formations."

In this instance the patient was not under Dr. Todd's observation at the time when the laceration may be supposed to have occurred. The patient, a man aged 21 years, was admitted into King's College Hospital, labouring under anasarca and ascites. The dropsy had commenced in the face and upper extremities, and had come on rapidly. The liver was much enlarged and indurated; the respiration rapid (40 in the minute;) the patient was troubled with a wasting cough, and he remained in the semi-recumbent position.

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[1] A Case of Rupture of the Chordæ Tendineæ of the Tricuspid Valve of the Heart, By Robert Bentley Todd, M. D., F. R. S. Dublin Quarterly Journal of Medical Science, New Series, vol. v.

Over the front of the chest the vesicular murmur was puerile, while posteriorly it was feeble and crepitant. The impulse of the heart was considerable, and it could be felt and seen over a large surface, with a thrill both at its base and apex. The impulse was to be seen and felt in the scrobiculus cordis and over the præcordial region. The sound was dull to a large extent. Two bellows murmurs, of different tone and intensity, were perceptible: one, loudest at the apex, and also distinct under the sternum. The second was less intense; its tone was different; it was audible at the base of the heart and along the course of the aorta. Both murmurs were systolic; and the second sound of the heart was natural, but feeble. The pulse was small, thready, and very compressible.

It appeared that his illness commenced at a period about two years and three months before his admission. He had been engaged in a riot in which he had received a stab in the right side, a little below the mamma. He bled freely from the wound, and was attacked with pleurisy, for the cure of which several venesections were practised. This attack was succeeded by costiveness, followed by the discharge by stool of a considerable quantity of fœtid clotted blood. Hæmatemesis set in about four weeks after the accident; and this symptom, as well as the discharge of blood by stool, recurred at about every fortnight up to the last three months of his illness.

I shall give the account of the dissection in Dr. Todd's words:—

"*Post-mortem Examination.*—The inspection of the body took place about twelve hours after death. The body was extremely œdematous; both pleuræ and the peritoneum were full of fluid, effused, as regards that in the pleura, during the last forty-eight hours. The lungs did not collapse; they were in a highly œdematous state. The heart presented considerable hypertrophy, with dilatation of the right ventricle and auricle; the left cavities were rather larger than the normal size; the valves of the left side were perfectly healthy, as were also those of the pulmonary artery; not so, however, the tricuspid valve, the anterior and largest division of which, that which separates the infundibular from the auricular portion of the ventricle, lay loose in the cavity of the ventricle; it retained its connexion with the heart only at its base, towards the auriculo-ventricular fibrous zone, inasmuch as all the chordæ tendineæ belonging to it were torn across at unequal distances, so as to give the free margin of the valve a ragged appearance; the musculi papillares, from which the chordæ tendineæ originally sprung, and to which portions of them still adhered, were distinctly shrunk and wasted; the margin of the valve was not thickened, nor could any morbid change be detected in any part of its structure, nor in that of the broken tendinous chords: the ends of these chords, both of the portions adherent to the valve, and of those attached to the musculi papillares, were swollen into little knobs, calling to mind the swollen ends of the nerves of stumps after amputation.

"The aorta and its branches were unusually small, and their coats much attenuated, so that they resembled in structure the pulmonary artery, which, on the other hand, partook of the dilatation and hypertrophy of the right cavities.

"The atrophied condition of the musculi papillares in the right ventricle, which are the regulators of the tricuspid valve, furnishes interesting evidence of the long existence of the rupture. Its date was probably to be referred to the time when the patient received the blow on his chest, a period of two years and a half. And the rounded and swollen extremities of the tendons indicated an effort at repair, which wanted for its success only the complete and continued apposition of the broken extremities, which, in such an organ, would, of course, be impossible.

"The mucous membrane of the stomach was extremely pale and thin, and here and there soft; there was no ulceration or cicatrix.

"There was nothing worthy of special notice in any of the other organs."

In the opinion of Dr. Todd, that the rupture of the tendinous cords took place at the period of the struggle during which the patient was wounded, we must agree. The musculi papillares corresponding to the broken cords were found shrunk and atrophied, proving that a considerable time had elapsed since the occurrence of rupture.

In considering the character of the symptoms which may attend this accident, as compared with those in which the mitral valves or their tendinous cords give way, Dr. Todd observes that the rupture of several tendinous cords, on the right side, would not immediately give rise to such serious symptoms as on the left. And he alludes to the natural imperfection of the tricuspid valve as illustrative of this principle. On this condition of the tricuspid valves I have already given the views of Dr. Adams and Mr. King; and we may admit, with Dr. Todd, that in consequence of the regurgitation from an over-distended auricle acting, in one case, on the lung, and in the other, on the great venous trunks, there would be a greater probability of sudden and severe symptoms being produced in the case of mitral than of tricuspid rupture.

Finally, the engorgement of the liver, and the frequent recurrence of hæmatemesis, are successfully traced by Dr. Todd to the overloaded state of the right auricle and venous system.[1]

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[1] "There was a combination of circumstances," says Dr. Todd, "in this case which rendered it extremely difficult to form a satisfactory and precise diagnosis. I, therefore, did not attempt one. The patient was so weak that frequent or minute examinations of him were impossible. I found, however, sufficient evidence of enlarged heart, in the extent of the dulness, and the increased force and extent of the impulse; and I came to the conclusion that the enlargement was due to hypertrophy and dilatation of both ventricles; the dilated condition of the right ventricle was sufficiently manifest from the extension of the heart into the region of the scrobiculus, where its beats could be distinctly seen and felt, and from the great extent of the dropsy, which is so apt to occur when there is dilatation of the right ventricle. The veins of the neck having been obscured by the anasarca in that region, the existence of venous regurgitation could not be satisfactorily ascertained.

The bellows murmur, so loud over the point of the heart, indicated imperfection of one or other of the auriculo-ventricular valves, probably of that of the left side, since it is more liable to a morbid state. It did not, however, escape my notice, that this sound was very distinct over the sternum, and that it possibly might be developed in the tricuspid orifice. But the rare occurrence of any lesion in that orifice, sufficient to develop bellows murmur, rendered such a diagnosis improbable, while the enlargement of the right ventricle, and probably of the left, and the extension of the former to the apex of the heart, would materially conduce to the propagation of sound generated in the mitral orifice, to the right side. The murmur in the aortic orifice was not necessarily indicative of any morbid lesion of that orifice, as the state of extreme anæmia which the patient exhibited was sufficient to give rise to it. Both bellows sounds were, no doubt, much increased in intensity by the anæmic state."

CHAPTER X.

DERANGED ACTION OF THE HEART.

The nervous diseases of the heart are practically divisible into two heads:—in one, so far as we can tell, organic or inflammatory disease is absent; while in the other, with a variety of structural diseases, deranged innervation is the predominating condition. Thus considered, the class of nervous affections of the heart is not only co-extensive with that of organic disease, but exceeds it by that category of cases in which we only find the evidences of a nervous state, original or sympathetic.

It is probable that uncomplicated neurosis of the heart is rarely met with; and the practitioner must not forget that, in a certain class of cases, where physical examination does not discover organic disease, the negative evidence as to the nature of the case is to be received with the same caution as in many other examples of disease. It is at all events certain, even though we be satisfied as to the absence of structural lesion, that marked derangement of action, in many cases, becomes a habit or custom of the heart in consequence of some influence of a sympathetic nature; or, as in the case of hysteric disease, the accidental localization of a wandering neurosis, which now affects one, and now another organ.

Angina pectoris has been described as a purely nervous affection. The researches of two illustrious observers, Heberden, in the eighteenth, and Latham, in the nineteenth century, have established, that whatever may be the immediate cause of the pain and sensation of approaching death, these symptoms are often met with in connexion with some form of organic disease of the heart. These forms have been indicated by Dr. Latham, and may be thus enumerated.

1. Weakness and attenuation.
2. Weakness with fatty degeneration.
3. Some form of valvular disease, generally affecting the left side.

4. Disease of the aorta, with or without obstruction of the coronary arteries.

From this comprehensive catalogue we may safely believe, that angina pectoris has occurred in association with most forms of disease of the heart. It is possible also, that not only local dilatation of the aorta, but other examples of aneurism, may be attended with symptoms of angina. To this point we shall refer when speaking of aneurism of the aorta. And we must admit, with Dr. Latham, that angina pectoris may be coincident with one, and one only, of these forms of disease, or with two or more of them in combination. It is greatly to be doubted, that angina pectoris has ever occurred in a patient perfectly free from organic disease of the heart or aorta; and it is more probable that, in the cases so described, the disease was overlooked, than that the heart was perfectly sound. Such cases as were observed before the application of the microscope to pathological anatomy, may be set aside, as proving the existence of angina without organic change,—for among the most important uses of histological research is the discovery of those early stages of organic change which escape the unassisted eye. On the possible absence of organic disease in this affection Dr. Latham expresses himself somewhat doubtfully:—"It has existed," he says, "where no form of disease or disorganization whatever has been found either in the heart or in the blood-vessels nearest to it."[1] But he gives no case of the kind as occurring in his own large experience.

In the present state of knowledge we must follow Dr. Latham, in considering angina pectoris rather as a special set of symptoms than a disease having a fixed anatomical character.[2]

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[1] Op. Cit. vol. xi. p. 362.

[2] The two concluding Lectures of Dr. Latham should be carefully studied, not only as bearing on the disease in question, but as fine examples of medical writing. On the point alluded to in the text he observes:—"Our knowledge, then, of angina pectoris stops short with its symptoms. The idea of it cannot be made to rest in any definite form of disease beyond them. We are sure of what it is as an assemblage of symptoms. We are not sure of what it is as a disease.

"There is a use sometimes in thus measuring the limits of our knowledge. In a profession like ours it is not enough to lament its imperfections. We should rather seek to understand wherein they consist, and to learn to bear with them and to make the best of them.

"Medicine is a strange mixture of speculation and action. We have to cultivate a science and to exercise an art. The calls of science are upon our leisure

It is difficult to define with accuracy the group of symptoms which constitute a paroxysm of angina pectoris. And, doubtless, we may have the condition of angina, properly so called, in connexion with other symptoms dependent on accidental combinations. In fact, an attack of angina pectoris in its greatest intensity, and at the same time simplicity, is of rare occurrence. Many cases denominated angina pectoris by one physician would be called cardiac asthma by another, and still the question remain open, whether in such instances there was not really a combination of two affections. We owe to Dr. Parry the best account of the disease in its simplest form, when it may be described as a paroxysmal affection, attended with pain in the præcordial region, generally propagated in the course of the nerves of Wrisberg, pallor of the countenance, and sensation of impending death. These terrible symptoms may supervene, and also subside, in a

and our choice; the calls of practice are of daily emergence and necessity. Science may minister to practice much or little. But whether science help us or fail us, whether its instrumentality be sufficient or defective, still we must act. We are bound to the constant endeavour of doing the best we can, whether upon a perfect or an imperfect knowledge.

"The imperfection of our knowledge, now especially pointed out, is, where all our pretensions to be exact must rest in the character of the symptoms, and cannot reach to the nature of the disease. But let us be just at least to such pretensions, and not run away with the notion that this knowledge is no knowledge at all. For the fact is far otherwise. Often, indeed, where there is much more knowledge besides this, yet is it this and this only that can be made use of; and all the rest goes for nothing when we come to seek for guides and indications of treatment.

"Think what symptoms are. They are not mere signs of the disease, but they are direct emanations from it; not things in themselves nugatory, but eminently real. They are natural sensations unduly exalted, or unduly depressed, or variously changed or perverted. They are natural functions hurt, hindered, or abolished. So that a man may often, with stricter propriety, be said to be ill of his symptoms than to be ill of his disease; and, what is more, to die of his symptoms than to die of his disease.

"Accordingly, it often happens, even where the disease is best understood, that we treat the symptoms, and the symptoms only, just as if we had no knowledge of any thing beyond them. Therefore, when we have confessedly no strict knowledge of anything beyond them, and the aim of our practice must needs centre in the symptoms, we are not to lament over the shortcomings of our art and its straitened capacity of doing good. For it does not follow that, if we knew the disease ever so well, we could treat it otherwise than we are now treating its symptoms, or that what we are now doing for the symptoms would not be the best, and would not be all, that could be done for the disease itself."

sudden manner; they may arise in a person apparently healthy; they are best relieved by stimulants and opiates, and, finally, as Dr. Latham has shown, the disease may prove fatal by repetitions of attacks within a short time after its first invasion, or even in its very first paroxysm.

If we inquire into the nature of a paroxysm of angina, two completely opposite views present themselves. Is it to be explained by spasm of the heart, or referred to diminished action? Dr. Latham, following Heberden, adopts the first of these doctrines, and has ingeniously defended it. Pain and the dying sensation are by him referred to spasm. "The paroxysm," he says, "of angina pectoris is plainly a compound of pain and of something else. Of the pain there can be no doubt. But there needs must be something more than the pain to account for the dying feeling which attends every paroxysm, and for actual death in a paroxysm at last."

"Spasm, it has been said, is a mode of action in muscular structure different from or beyond the natural or accustomed mode. The natural actions in all muscles, voluntary and involuntary, are unaccompanied by any conscious sensation whatever. But spasm is always accompanied by pain, and pain and spasm, wherever they are, disable the parts which they befall. Colic stops the peristaltic action of the bowels. Cramp forbids the hands to handle, and the feet to walk."

"But the heart is a muscle, and its functions flow from its attributes as a muscle. Now we are in search of something in the heart which, as the concomitant of pain, may be disabling to its natural functions, and capable, according to its degree, of hindering or abolishing them altogether. This we find in spasm. In its spasm of smaller degree the heart fails to close freely upon the blood, and to impel it freely into the arteries. In its spasm of greater degree, it fails to project it altogether."

"Hence we discern an adequate explanation of the chief phenomena of angina pectoris: it is a spasm of the heart."[1]

Finally, Dr. Latham instances the efficacy of opium in relieving the patient. The success of this medicine, in removing those muscular spasms which occur in cases of fracture, might be quoted in favour of his view of the matter.

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[1] Op. Cit. p. 885.

Yet before we assent to the doctrine of spasm, some general considerations are necessary. Looking at the class of patients who are most liable to angina pectoris, we find them generally persons advanced in life, and often of a full or leuco-phlegmatic temperament. They may be the subjects of atonic gout; they frequently suffer from hepatic disturbance, and, in many instances, present signs of valvular disease, with or without a diminished muscular energy of the heart. It is in such persons that a weakened heart, either from attenuation or from fatty degeneration, is most commonly produced. And it appears certain, that angina is rare in active hypertrophy. If we place the cases of weakened heart, from any cause, in one category, and those in which the muscular energy is either not deficient or in excess in the other, we find a much greater number of cases of angina in the first than in the second series.

Bearing in mind that the heart may be considered as a hollow muscle, it is difficult to understand how such a general or local spasm could occur as would only impede, and not destroy, its function; for a complete spasmodic closure of any one cavity ought to cause death by breaking the continuity of the circulation. In such a case, too, we might expect to find after death that the heart, or a portion of the heart, was firmly contracted on itself. Yet, so far as I know, such a condition has not been found in persons who have died in a paroxysm of angina. Little as is known of spasm of the heart, that little is opposed to the idea of angina pectoris being produced by it. I have already noticed the spasmodic contraction of the heart in tetanus, and in such cases the symptoms of angina were not observed.

In examining this question the following considerations must not be lost sight of:—

1. That angina pectoris has seldom been found without organic disease affecting the heart or arteries, and that in the same individual there may be various forms and seats of disease.

2. That the individuals most liable to angina are those in whom we find some form of weakened heart, the evil effects of which will be augmented by associated organic diseases, engaging the muscular structures, endocardium, valves, coronary arteries, or the aorta itself.

These considerations lead to the doctrine long since indicated by Parry, that the symptoms of angina arise from a temporary increase of weakness in an organ already weakened.

The views of Dr. Parry upon this vexed question are opposed to the doctrine of spasm of the heart. Putting aside his opinions as to the obstruction of the coronary arteries, a condition which, though frequent, is not necessary, we find that he refers the symptoms and fatal termination of the disease not to any increase, but to a diminution of the muscular power of the heart. The disease is, according to him, an example of syncope, preceded by a notable anxiety or pain in the region of the heart, the result of organic lesion, which acts in diminishing the energy of the heart; and he holds, that the symptoms arise from the retardation and accumulation of blood in the cavities of the organ.[1]

Dr. Parry has insisted strongly on the absence of dyspnœa, and, indeed, of palpitation, in the best-marked cases of this disease. The patient, it is true, may present accelerated breathing, or some form of that sighing respiration already noticed in the chapter on fatty degeneration of the heart, and it is probable, that these conditions have been described as dyspnœa. But it is certain, that the extreme difficulty of breathing which we observe in cardiac asthma forms no essential part of the paroxysm of angina pectoris. Heberden observes that, with the exception of the pain affecting the breast, the patients are at the beginning perfectly well.[1] And in the cases met with by Parry, and those communicated to him, the patients were so far from suffering dyspnœa, that they frequently made deep inspirations, and retained their breath not only without reluctance, but with much pleasure. It is evident, therefore, he adds, that in these cases there was neither difficult nor hurried respiration. In a case recorded by Parry, the sufferer was in the habit of resting on a full inspiration, as it afforded a momentary relief to the uneasy sensation in the chest; and Heberden observes, that the patients obtain ease "by straightening the vertebræ of the thorax."

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[1] Dr. Parry's work, entitled, "An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris," and published in 1799, is unquestionably the best monograph we possess on this subject. In his recapitulation (see page 141) he observes, that the causes exciting the paroxysms are those which produce accumulation of blood in the heart's cavities, and that this is effected by mechanical pressure or by over-stimulation of the circulating system: "in consequence of which, the heart, weakened by the mal-organization, readily sinks into a state of quiescence, while the blood continues to advance in the veins. Whence it follows, that the power of the heart being given, the disposition to paroxysms will be directly as the momentum of the blood in the veins; and that, on the contrary, the momentum of the blood in the veins being given, the disposition to paroxysms will be inversely as the power of the heart." And he adds, that after a certain approach towards quiescence, by which he means suspension of action, the heart may recover its irritability so as to carry on the circulation in a more or less perfect manner, but that death may at last ensue, to use his own words, "from a remediless degree of inirritability in the heart."

In fact, the respiratory phenomenon which belongs to angina is some form of the sighing respiration, so important a symptom in the fatty or weakened heart, and its occurrence is an additional argument against the doctrine of spasm; for this symptom, taken in connexion with the almost imperceptible pulse, which is commonly observed during the attack, points too plainly to a weakened, semi-paralyzed state of the heart.[2]

Upon the whole, we may conclude, that the special group of symptoms described as angina pectoris by Heberden, Parry, Percival, and Latham, is but the occurrence, in a defined manner, of some of the symptoms connected with a weakened heart. Obstruction of the coronary arteries may or may not be present, and is probably not unfrequent; but, as a cause of angina, its action is remote, and its existence unnecessary. It is only by causing atrophy with fatty degeneration, as Dr. Quain has shown, that it appears remotely to produce angina, a condition which might arise from similar states of the muscular structure, even though the arteries remained pervious.

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[1] Medical Transactions, vol. iii. p. 3.

[2] We might inquire whether, under these circumstances, there is not some consent between the heart and the lung itself. The alterations of high suspirious breathing with periods of apnœa, as noticed in the fatty disease of the heart, indicate derangement of the pulmonary nerves. Are the forced inspirations induced by an effort to carry on circulation by the sucking-pump action of the thorax? And do the apnœal periods indicate a condition of the lung corresponding to the temporary paralysis of the heart? We might further inquire whether, in such cases, there may not be an actual degeneration of the muscular structures of the lung similar to that of the heart, and presenting, under a chronic form, that co-existence and similarity of disease which we have hinted at as probable in the secondary affection of typhus fever.

If the views of Abercrombie as to the nature of ileus be adopted, we may, by the use of analogy, throw some light on this question. In this disease we find weakness or paralysis of a muscular tube; but this is partial, for above and below the dilated portion the muscles retain their power. If a partial weakness of the heart was to occur,—a condition much more likely to arise than a partial spasm or augmentation of contractile force,—we would have in the heart a mechanical condition not very dissimilar to that in the intestinal tube when affected with ileus. If the left ventricle was the seat of the paralysis or temporary weakness, while the right continued to act, obstruction and dilatation would equally follow, and if the loss of power in the left ventricle was complete, syncope, or, it might be, death, would be the result.

What has been now said, however, must be taken less as an argument against the possibility of spasm of the heart than as an attempt to prove that angina pectoris proceeds from an opposite condition. We might explain the pain from the over-distention of collateral vessels; and the syncope is as easily understood on one hypothesis as the other. But let it be remembered that, while the proofs of syncope arising from a weakened or paralysed heart are sufficiently abundant, its occurrence from a spasmodic condition is by no means established.

In considering this subject we must not forget, that under the term angina pectoris, physicians have included, and still include, many examples of diseases which vary in their nature and combinations. Well-marked instances of the affection, as described by Dr. Latham, are rarely met with, and the same may be said of the purely nervous cases noticed by Laennec. I have never seen either of these forms. The disease which in this country most often gets the name of angina pectoris might be more properly designated as cardiac asthma. It is probable, that this is an example of the combination of a dilated heart, with congestion and spasm of the lungs. This theory would, at least, explain the symptoms in many cases of cardiac asthma, especially when arising suddenly in individuals whose hearts were weakened and permanently irregular.

Some useful information might be supplied by accurate observations upon the following points:—

1. The physical phenomena of the heart, as occurring during an attack of angina pectoris. The state of the pulse should be noted, and the impulse and sounds carefully examined at three points, viz., the base of the heart, the left mammary region, and the xiphoid cartilage.

2. Comparative observations of the heart's sounds and impulses during the intervals of the attacks.

3. The determination of the state of the cavities of the heart in persons who have died in a paroxysm of angina.

4. The determination of the amount of blood found in each or any of the cavities.

5. The condition of the aorta should be carefully examined, not only with reference to the discovery of the more ordinary diseases, but to determine how far its elasticity has been injured. And again, whether or not a dissecting aneurism may have existed. This disease is often overlooked unless when hemorrhage into the pericardium has occurred.

Treatment.—I have nothing to add to the treatment of the paroxysm of angina as given in systematic works on the practice of medicine. Stimulants are, by general consent, looked on as the principal remedies. Of these, wine, brandy, ammonia, and Hoffmann's ether, are those on which we must rely, the quantity exhibited being regulated by the effect. Mustard poultices and hot fomentations may be applied to the præcordial region. And it is necessary to remark that, in the management of these remedies, we are to be guided not only by the general state of the patient, but by the careful examination of the impulse and sounds of the heart. Further, if by percussion we find that the stomach is distended with flatus, we must employ appropriate remedies to relieve the part.

For the relief of the pain Dr. Latham relies on opium. He advises the exhibition of from thirty to sixty drops of laudanum. We may derive some guide, in the use of this and other anæsthetics in the treatment of angina, by our previous knowledge of the force of the heart's action. And where there is no reason to believe that the heart is very weak, we may, perhaps, be more bold in employing this treatment. Recent experience seems to show that in the case of fatty heart there is danger from the inhalation of chloroform. It may be, however, that the exhibition of a draught containing ten or fifteen drops of chloroform might act beneficially. I may here mention a clinical fact, which I owe to a distinguished practitioner in England:—A lady, of a nervous temperament, who had long suffered from intercostal neuralgia at the left side, consulted him. He applied chloroform over the præcordial region, and in a few minutes a violent convulsive attack supervened, followed by a long collapse, which required the exhibition of stimulants for many hours.

The general treatment of patients liable to attacks of angina pectoris must be that adapted to cases of the weakened or fatty hearts.

In considering the functional derangements of the heart, we may adopt the practical division of the cases into those in which the palpitations, pain, or other symptoms, occur independent of any disease external to the heart; and those where they are apparently excited either by sympathy with various local affections, or a generally deranged state of the system. Yet, even if we adopt this simple arrangement, we find many cases of so mixed a nature that they cannot justly be placed in either category.

In place of bewildering ourselves with difficult or impossible classifications, let us discuss, in a clinical point of view, some of the more common cases which come before us.

Simple Neuralgia of the Heart.—A pain, referred by the patient to the heart, and it may be attended with palpitation, is often complained of. In some cases the latter symptom is not necessarily attendant on the pain, but is really induced by the apprehensions of the patient. It is very difficult to distinguish true neuralgia of the heart from some of the common forms of intercostal pain, and, in most cases, the supposed cardiac neuralgia is not a symptom of consequence. Indeed, pain in the heart is by no means a common attendant on the early stages of its chronic diseases, which, in so many instances, begin silently and painlessly. If, however, we find that the pain is obstinate,—that it is attended with palpitations which do not proceed from apprehension,—if it affects the left upper extremity, and cannot be found to depend on gastric derangement,—we must not neglect it, nor allow even the complete absence of physical signs of disease to make us too confident that some obscure lesion of the heart or aorta is not in progress. In this diagnosis we should, of course, carefully weigh the age, habit, and temperament of the patient. And further, we are not to allow ourselves to conclude absolutely against the existence or the threatening of organic lesion because the pain is inconstant; for intermitting pain may occur in various forms of steadily progressive disease.

In connexion with this subject, the following case is at least instructive:—I was consulted by Dr. Lyons in the case of a gentleman, aged about 57, who had for some time suffered from general malaise and gastric derangement. He also complained of a strange sensation of heat in the left arm and extending up the side of the neck. Indistinct sensations of the same kind were felt in the left lower extremity. At this time the most careful examination failed to discover any disease of the heart. The symptom in question had been of about two years' standing. In a year subsequently I saw him again, the symptoms having continued. The phenomena of the heart were still natural.

In this state he continued for three years, the sensations of heat in the arm being subject to occasional aggravation, when he suffered from an access of muscular rheumatism. Six months subsequently, on a casual examination by his attending physician, a bellows murmur, having its seat apparently in the mitral opening, was discovered. In about a year afterwards the patient had an attack of pericarditis, and it was found that during the greatest intensity of this disease the mitral murmur disappeared. It, however, returned on the subsidence of the pericardial sounds, and has since continued with the addition of signs of hypertrophy of the heart.

In the interpretation of the symptom of heat in the left arm, it must be mentioned that the patient, after the age of twenty-five, was attacked with epilepsy, which recurred for about five years, and he has occasionally suffered from vertigo, but up to the present time no other symptom of any cerebral disease has ever shown itself.[1]

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[1] The discovery by Remak of the existence of microscopic ganglia on the surface of the heart, similar to those which he has found to exist in the bronchial tubes and the stomach, must be noted here. The application of this observation to practical medicine is yet to be made. (Müller's Archives, 1852.)

The occurrence of neuralgic pain in the arm or shoulder is a circumstance which should always excite attention, for it is commonly a symptom of some form of intra-thoracic disease. The pain, especially where it depends on cardiac disease, appears to follow the course of the nerves of Wrisberg, and may be propagated to the fingers. It may be paroxysmal, intermittent, or permanent, and it is often relieved by external anodyne or stimulating applications, or, when it is intermittent, by bark. This circumstance frequently leads to a misapprehension of its nature, and in this way various important diseases, of which it was for a time almost the only manifest symptom, are often overlooked. We may thus see cases of aneurism, various forms of chronic disease of the heart, the early stages of tubercle of the lung, some forms of pericarditis, and, I think, cases of cancerous tumours within the thorax, in which this pain has been an early, though often misinterpreted symptom.

In the treatment of cardiac neuralgia we should expect benefit from all measures that improve and fortify the general health. Should gouty or dyspeptic symptoms be present, these must claim especial attention. A graduated course of exercise will often have the best effect, and the remedies which are in use in neuralgic diseases generally may be employed. The proto-carbonate of iron is often useful, given with or without some aromatic; and the aromatic iron mixture of Heberden, in combination with laurel water, or the diluted hydrocyanic acid, is a convenient remedy. Among the external applications we may place belladonna, aconite, and veratria, in the form of liniments or plasters. I have not employed the electro-magnetic current, but from its efficacy in other forms of nervous disease, it will probably be found efficacious. But one of the most certain remedies consists in removing from the patient's mind the apprehension that his heart is organically diseased.

NERVOUS PALPITATION.

This condition is met with under an infinite variety of circumstances. In its simplest form we observe nothing beyond a natural susceptibility or excitability of the heart, which we cannot connect with any anatomical change of the organ, or sympathy with disease in other parts. Some individuals, then, have naturally an excitable heart, ready to be affected by palpitation, under a great variety of circumstances.

In the next place, we observe nervous palpitations coincident with or dependent on special conditions of the system.

Some of the most singular examples are met with in hysteria, especially in that form where the nervous disturbance engages many organs successively or simultaneously.

The palpitations which occur from anæmia are examples of excited action, dependent on a general condition. And we may place in the same category the cases already alluded to, of non-inflammatory excitement of the heart in the advanced stages of typhus.

Another important series of cases is that in which the heart's action is excited and irregular, apparently from the action of some organic sympathy. A common example is the union of hepatic and cardiac symptoms already described. I think that in such cases, at least in the first instance, the irregular action of the heart is often purely sympathetic. And I apprehend that we may have a case of enlarged and otherwise diseased liver, with excited and irregular action of the heart, in which, especially where the disease does not occur in advanced life, the period of change from a merely functional to an organic lesion of the heart cannot be determined with accuracy. For, under these circumstances, the physical signs of a heart which has passed into dilatation, and of a heart which may unexpectedly resume all the characters of natural action are closely similar. Theoretically, we should be assisted by percussion in the diagnosis between these cases, but in practice this method is often fallacious. And again, it is not impossible that, under functional disturbance, we may have a temporary dilatation of one or more of the heart's cavities, so as to affect the results of percussion.[1]

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[1] See the chapter on "Dilatation of the Heart." The practitioner cannot be too careful in committing himself to a diagnosis of organic disease of the heart in certain cases of the combination of cardiac and hepatic symptoms, especially when the disease exists in persons not much advanced in years. For we shall see that under treatment calculated to relieve the liver and digestive system, the symptoms and signs of the dilated and irregular heart may suddenly disappear; and this, too, while an enlarged state of the liver still exists. In these cases murmur is rarely if ever present, the signs being an extremely irregular action of the heart, with clear and extended sounds, jerking impulse, and corresponding pulse.

I have already given two cases in which an extreme degree of palpitation and irregular action seemed to depend on some condition of the stomach, which was removed by vomiting. In one of these cases the patient was supposed to labour under acute carditis, and in the other, under complicated organic disease of the heart.

Let us, for the sake of clearness, enumerate the cases of nervous palpitation which have been briefly sketched out:

1. Simple nervous palpitation, unconnected with any general pathologic state.

2. Nervous palpitation, dependent on, or coincident with, a general morbid state. The following examples are common:—

a Hysteric palpitation.

b Gouty palpitation.

c Irregular and excited action, dependent on anæmia.

d Excited action of the heart, often observed in the advanced stages of essential fevers. This condition probably reveals in the heart a state analogous to that of the brain in typhus, when we meet with delirium, convulsions, and other symptoms, a state on which dissection throws but that negative light, telling, not what it is, but what it is not.

I have never found evidence of any form of carditis in these cases; and in their commonly fatal result we only see another exemplification of the rule, that in fever the excess of nervous symptoms should lead to a bad prognosis.

Lastly, we have those cases of nervous palpitation in connexion with disturbance of the gastric and hepatic functions. Here the symptoms seem to depend on some local organic sympathies. In both cases it may be long continued, and, where the liver is in fault, exhibit remarkable remissions. Sooner or later the heart becomes organically changed, and is weakened, dilated, and perhaps hypertrophied. But, as has been before stated, it appears impossible to mark with exactness the period when the excited and irregular action which, for a length of time, has merely indicated functional disturbance, becomes a symptom of an actually dilated and otherwise altered heart. The difficulty which attends the formation of a correct diagnosis in these cases is much greater than is supposed. It has happened often to me to find the action of the heart, which, for many months together, had been in the highest degree irregular, suddenly restored to a condition in which the rhythm and sounds were perfectly natural. And I have been informed by the medical attendants of such patients, that the state of excitation and irregularity of the heart had dated many years back, with occasional intervals of natural action. We as yet know little of the laws which govern the supervention of organic disease on functional derangement of the heart; but it is probable that in a case where there is no disposition to the fatty degeneration, and where the valves escape injury, a much longer time than is supposed must elapse before change in the muscular structures follows on nervous palpitation.

Another illustration of this doctrine is to be drawn from the return to a normal condition of the heart in those cases of long-continued violent action which are attended with the enlargement of the thyroid gland and eye-balls. I have already expressed my opinion, that the essence of this disease is a nervous state of the heart.[1]

The facility of making a correct diagnosis between functional and organic diseases of the heart is not so great as modern writers lead us to believe. And we more often arrive at a just conclusion by instinctive skill, the result of experience and judgment, than by communicable rules of diagnosis.

Considered with reference to the facility of diagnosis, the cases of nervous palpitation may be practically divided into two classes. In one there is regular, in the other irregular, action of the heart. In either of these cases inorganic murmur may or may not be present. I think its occurrence is more frequent with the first than the second variety; and yet the diagnosis, where the heart is acting regularly, is less difficult than in cases of an opposite description.

If we inquire whether there is any acoustic character by which murmurs, not depending on organic disease of the heart, can be safely distinguished from those of the opposite description, the question must be answered in the negative. Dr. Hope, indeed, holds, that the nervous murmurs are always of a soft blowing character; but this opinion is too positive, for every practical man knows that, both in the heart and aorta, organic murmurs may be low and soft; and that inorganic murmurs are occasionally harsh, loud, or musical.

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[1] See chapter III. page 308.

The diagnosis is to be made by a careful consideration of the following points:—

1. The previous history of the patient.
2. The existing vital symptoms.
3. The duration of the murmur.
4. The amount of accordance between the physical signs, supposing them indicative of organic disease, and the history, general state of the patient, and condition of the cavities of the heart, so far as this latter can be ascertained.
5. The state of the heart, as to general excitability, is important. We do not say that disease of the valves is never attended with excitement of the heart's action, nor that inorganic murmur may not co-exist with a quiet heart; but this is certain, that we much more often meet with quiescence in connexion with disease of the valves, than in cases presenting the inorganic murmurs. Murmur, then, wherever it may be seated, co-existing with a tranquil and regular action of the heart, is more likely to proceed from organic disease than the opposite. This is more common when the murmur is seated in the mitral than the aortic orifice.
6. The seat or the point of greatest intensity of murmur must be, if possible, determined. Drs. Hope and Walshe have concurred in the statement, that its seat is at the arterial orifices. Dr. Hope says:—"It is confined to the aortic orifice (so far as I have yet discovered,) and to the first sound;" and Dr. Walshe observes, "that, according to his experience, it is invariably basic in seat, and systolic in time." If these opinions be accepted, the diagnosis would stand thus:—That a systolic aortic murmur, unattended with the symptoms and remaining signs of organic disease of the valves, especially those of regurgitation, should be considered as inorganic.[1]

But we must be cautious in rejecting the opinion, that inorganic murmurs may not be seated in the mitral orifice. As a general rule, we may adopt the statements of Drs. Hope and Walshe; but I cannot help believing, that I have observed cases of inorganic murmurs which, so far as physical signs went, were closely similar to those of ordinary mitral disease with regurgitation into the auricle. And it must be borne in mind, that we are under difficulties in determining that a murmur is only produced at the base of the heart when the concomitant signs and symptoms which attend disease in that situation are wanting. Again, cases may be met with in which the action of the heart is so tumultuous and irregular as to make the determination of the seat of murmur practically impossible.

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[1] The question of disease of the pulmonary valves may be neglected in dealing with practical diagnosis.

We may, however, admit, that in most cases of inorganic murmur, the sound is produced during the systole; it is occasionally propagated into the aorta, and may be attended with murmur in the jugular veins, or, as some assert, in the venæ innominatæ. Confining ourselves to physical signs, the diagnosis of anæmic murmur of the heart will often stand as follows:—

1. A murmur, coincident with the first sound of the heart, generally louder at the left than the right side.
2. A clear second sound.
3. Murmur in the course of the aorta.

Adding other considerations, we arrive at the following rule, which is applicable in many cases, viz., that in a patient whose age, general appearance, and history, are opposed to the supposition of chronic organic disease; in whom the pulse has not the character of regurgitation; and the signs of hypertrophy of the left ventricle are absent,—the existence of a single murmur with the first sound, generally predominating towards the base, and accompanied by an aortic murmur, while, at the same time, the second sound is clear,—should make us conclude that the signs proceed from anæmia.

In most cases the murmur has the soft, blowing character described by Hope, but in others it is decidedly musical, and varies in its tone from time to time.

I have never met a case of mere anæmic or nervous murmur, in which the second sound became masked, as in permanent patency of the aortic valves; hence the absence of murmur with the second sound is of great value in this diagnosis. But we must here notice a form of disease calculated to mislead the inexperienced practitioner. It is the co-existence, in a young person, of regurgitant disease of the aortic valves with the anæmic condition. I have never seen this disease in a female, but have observed it in the male between the ages of fourteen and twenty. In such cases the character of the pulse, especially when the heart is excited, and the masking of the second sound by regurgitant murmur, are the circumstances which should prevent us from concluding that the cardiac phenomena depended solely upon anæmia. It is important to bear this in mind, inasmuch as we find the anæmic condition at any age, but especially in the young, to co-exist more frequently with disease of the aortic than of the auriculo-ventricular valves.

But, on the other hand, to state that the triple combination of a murmur with the first sound,—a clear second sound,—and yet murmur in the aorta, warrants the diagnosis of a purely anæmic condition, would be erroneous; for, as has been already shown, this very combination exists where the disease of the aortic orifice is such as, while causing irregularity of surface on the ventricular side, it yet permits the valves to close at the arterial systole. Up to the present time I have only met this special condition in cases of fatty degeneration of the heart occurring in advanced age; hence it may be stated, that the triple combination in question occurs but in two cases, one, the anæmic or chlorotic condition, commonly seen in young persons; the other, the fatty state of the heart with disease of the aortic opening, generally met with in advanced life. The practical man will have no difficulty in distinguishing between these cases.[1]

It would greatly advance our knowledge of this part of medicine if an extended series of observations was made upon the combination of organic and inorganic murmurs. We should determine how far an inorganic murmur modifies the tone and character of murmurs of valvular disease; and again, whether an anæmic murmur at one orifice can be at first distinguished from an organic at another. Cases of this sort are more frequent than is imagined; and their diagnosis, even to the best-informed physician, is full of difficulty. Let us suppose a case of regurgitant disease of the mitral opening, with its proper sound, in which anæmic murmur exists at the orifice of the aorta,—one of these murmurs may be louder than the other; one or both may be musical; both are systolic; and fremitus may or may not be present:—he would be a bold and a badly-informed physician who, on a first examination, would declare positively in which orifice the disease was seated, or, indeed, whether organic disease existed at all. Doubtless, the advance of diagnosis will diminish these difficulties, but it is most desirable that the younger practitioner should not enter on his profession believing that as he runs so he may read the varied signs of cardiac disease. Let us refer to a case, already mentioned in this work, which illustrates what has now been said:—A young lady, of extreme beauty and intelligence, had for several months laboured under the symptoms of chlorotic anæmia, attended with palpitation, dyspnœa, and œdema of the feet. There was no evidence that she had ever had carditis or any form of rheumatism, and the symptoms in question supervened upon a greatly diminished uterine action.

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[1] See the chapter on "Fatty Degeneration of the Heart."

The physical signs were as follow:—

1. The sound, on percussion over the heart, was natural.
2. The systolic sound was masked by a loud musical murmur.
3. The second sound was perfectly healthy.
4. A loud musical murmur existed in the aorta, subclavians, and carotids.

There were no signs of pulmonary congestion, and it was determined to treat the patient for anæmia, as its existence was sufficiently clear, while that of organic disease was doubtful. This treatment, after some time, seemed to be perfectly successful, for all the symptoms of heart disease subsided, and in the course of a year this lady was restored to her former health and beauty. She could walk up hill, ride, and dance with pleasure; and the lividity of the lips, which had been very remarkable, completely disappeared.

As to the physical signs, the following changes were observed: first, the jerking and vehement pulsation of the heart diminished; next, the murmur disappeared from the arteries, beginning with the carotids; next, the loudness and musical character of the cardiac murmur ceased, until nothing was left but a greatly diminished and softened systolic murmur, the second sound always remaining clear. It was then believed by several of her medical attendants that all her former physical signs had been simply anæmic, and that her health, in fact, was perfectly restored. Such, however, was not my opinion, for I felt uneasy at the continuance of this cardiac murmur, which, had it been of the same nature as that in the arteries, would have equally subsided on the removal of the anæmic condition. About two years subsequently I met this lady, and took an opportunity of examining her heart. She was then, to all appearance, in flourishing health. I found the usual signs of mitral disease, the action of the heart being perfectly regular; the murmur had lost all musical character, and had some of the roughness observed in cases of irregular ossification. Of course, care was taken not to convey a suspicion of the actual state of things to the lady. She continued in perfect health for another year, when, without any previous warning, she suddenly died. There was no dissection.

I have dwelt on this case as illustrating the combination of organic with functional disease of the heart. There can be little doubt that organic disease of the mitral valve preceded or at least accompanied the anæmic phenomena; and it is important to observe that the symptoms of cardiac suffering, the œdema, palpitation, dyspnœa, and debility, all disappeared on the removal of the anæmic state, furnishing another illustration of the great principle that, within certain limits, the sufferings of organs depend more on their vital than mechanical conditions. We must believe that the weakness and irritability of the heart was induced by the anæmia, as, when this was removed, its functions were carried on, in a healthy manner, for nearly three years.

The removal of the musical character of the cardiac murmur was very remarkable. Various authors have noticed the influence of anæmia in modifying the sound in valvular disease, but the subject requires additional investigation. Was there in this case a musical character given to the mitral murmur? Were there two murmurs, both systolic, one musical and the other not; the musical murmur at the base of the heart masking the ordinary mitral sound by its louder tone? or was the case one in which all cardiac murmur was confined to the mitral opening, though modified by that anæmic state, from which arose the arterial murmur, while the second sound remained unaffected?

Another example has been given (see Case XVIII. p. 167) of well-marked chlorotic anæmia in a young woman. The symptoms were of considerable standing. A distinct murmur was found at the left side of the heart; the second sound was healthy, and there was no arterial murmur. While this girl was under observation, we could not satisfy ourselves as to the existence of organic disease, and she was treated for the general affection. She died within two years; and her heart was exhibited at the Pathological Society by Dr. Bigger, who had charge of the patient before she had entered the Meath Hospital. The left ventricle was slightly hypertrophied, but the mitral opening was extremely contracted by union of the valves; the left auricle was dilated and hypertrophied.

This case illustrates the difficulties which attend on the diagnosis; and I doubt much whether, if a similar set of circumstances were presented to me, I could arrive at a more correct conclusion. Had arterial murmur existed, the signs would have been the same as in the preceding case. Again, it shows of what importance a knowledge of the previous history is in this condition. In Dr. Bigger's communication he stated that, before the patient entered hospital, she had gone through an attack of pericarditis. Of this we were not aware; otherwise there would have been less difficulty in the diagnosis.

Finally, the co-existence of venous murmur, at one or both sides of the neck, is not conclusive as to the character of cardiac murmur. It is only valuable as a sign of the anæmic condition, which may or may not co-exist with organic disease.

OCCURRENCE OF MURMUR IN FEVER.

We have already seen that, although the occurrence of murmur, from any cause, is rare in typhus fever, even when the heart has been softened, yet, during the last few years, it has been observed at some period of the short relapsing, or so-called typhoid fever of this country. A case has been given in a well-marked example of maculated typhus. In some of the typhoid cases there was reason to believe that the murmur might have arisen from an anæmic state existing previous to the fever; but in many others it was clear, that an anæmic murmur was produced in consequence of the fever itself. To this sound may be given the name of the typhoid anæmic murmur, and as its history is closely connected with the subject now in hand, and as the murmur itself is often accompanied by other signs referrible to functional lesion, no apology is necessary for returning to its consideration. The observations now to be made must be considered as supplemental to those already given in the chapter on the state of the heart in typhus fever. All these cases occurred in the Meath Hospital, and were noted by my friends, Dr. Heslop, of Birmingham, and Dr. Lyons, of this city.

CASE LXIX.—*Production of a Murmur during convalescence from Maculated Typhus; Persistence of this phenomenon during the subsequent relapse.*

A girl, aged 13, who had gone through a maculated typhus fever, of fourteen days' duration, presented the following signs after the first week of her convalescence:—The first sound of the heart was loud and prolonged, but when the patient sat up, this prolongation disappeared for six or seven beats, and then became again audible. In the course of three days she appeared to relapse, and had an attack of fever, which lasted for about ten days; there were no maculæ, and during this access the cardiac phenomena continued, the prolongation of the first sound passing into bellows murmur, while the second remained sharp and clear. This attack subsided, and the pulse again fell to the natural standard, but the morbid signs of the heart remained. An ephemeral relapse occurred within a week, after which she rapidly convalesced; and was soon discharged from hospital, the abnormal cardiac signs having completely subsided.

We have already noticed the greater frequency of this form of murmur in the typhoid than in the typhus fever; and it is worthy of remark, that in this case the murmur preceded the relapse, which, as is often observed, had a well-marked typhoid character, although the primary attack was one of maculated typhus. The prolongation of the first sound of the heart, whatever be its cause, is closely related to, though certainly different from, inorganic murmur. It is true, that the same general causes seem to give rise to both. It appears to arise from irregular contraction of the muscular fibrillæ. In some cases, especially in thin persons, we have found it attended with a peculiar vermicular sensation to the touch, very different from the fremitus of organic valvular disease. To this point we shall return. Like the anæmic murmur, it subsides during or after the convalescence of the patient, but I think that, when well established, its ultimate disappearance occurs much later than that of a true murmur.

But for the production of this sign, after a maculated typhus, it is not necessary that symptoms of the short relapsing fever should then exist. It occurred in a well-marked form in a boy aged 19, who recovered from a severe typhus fever with profuse maculæ, and typhoid consolidation of the lung. On the nineteenth day the pulse had fallen to 52 in the recumbent position, with an intermission at every second or third beat; but in the upright position the pulse rose to 92, and became regular and stronger. The prolongation was observed in the recumbent position, particularly during the throb preceding the intermission; the sound was most intense at a point midway between the sternum and left nipple. When the patient sat up, the prolongation altogether disappeared; in a short time it wholly subsided, and he left the hospital, restored to health.

During the years 1847 and 1848, similar phenomena were observed in the non-maculated typhoid fever, the point of greatest intensity of sound being midway between the nipple and sternum, and the sign disappearing or diminishing in the upright position.

These facts, with those recorded in our seventh chapter, prove the existence of a special form of inorganic murmur in connexion with typhus, but more especially with typhoid fever. If we now suppose a patient presenting this form of murmur to be seen for the first time, our diagnosis will be facilitated by attention to the following circumstances:—

First, the fact of the patient being at the time in typhus fever, which is by far the rarest case;—in the convalescence from typhus fever;—in those false convalescences, or, more properly, the short intermissions of the relapsing fever; or lastly, in the typhoid disease itself.

Secondly, the nature and seat of the murmur are to be considered; it is seldom musical, but has the soft blowing character commonly observed in anæmic murmurs, and its point of greatest intensity cannot be referred either to the aortic orifice or to that portion of the chest where mitral murmurs are supposed to be most intense.

Thirdly, its frequent disappearance or diminution in the erect position. This may help us in distinguishing it from an organic murmur, but I am unable to say whether the same is not observable in ordinary cases of anæmia.

Fourthly, the prolongation of the muscular sound, closely related to, and sometimes passing into, the anæmic murmur, is to be noted. Both phenomena appear to be governed by the same general conditions.

Fifthly, the rarity of carditis in typhus or typhoid fever is to be borne in mind, and this, together with the frequently rapid subsidence of the murmur, and absence of symptoms of cardiac inflammation, will, when taken with the preceding considerations, enable us to come to a just conclusion. It is true, that cases arise in which the differential diagnosis is full of difficulty, part of which, however, may be attributed to our ignorance of the previous condition of the patient. I have lately observed an instance of the difficulty which this diagnosis sometimes presents. A man, aged about 30, was convalescing from a protracted non-maculated fever, when, within a period of three days a soft systolic murmur became developed to the left of the nipple, and as there had been no symptoms of carditis, the case was set down as an example of the typhoid murmur. Within a few days, while examining this patient's lungs, I found an interscapular murmur considerably louder than that in the heart, and precisely similar to the murmur in extreme cases of mitral regurgitation in young persons. The patient's convalescence was progressive, and I kept him in hospital for a long time after his restoration to health, in the hope of coming to some satisfactory conclusion, but the signs remained unchanged, the heart's action being perfectly quiescent, and the man at length returned to his occupation, the sounds having been in no respect altered.

How are we to interpret this case? The signs appeared during an epidemic, in which the cardiac murmurs were common and the fever was of the character most likely to induce them. Was it an example of unusual persistence of the cardiac murmur, attended with some form of that localized murmur in the descending aorta, which is found in certain cases of nervous or chlorotic disease? For it is to be observed, that neither in the course of the ascending aorta nor the arch did this singular sound occur. Again, might the case have been one of long-existing mitral patency, in which, during the temporary weakness of the heart, the murmur subsided to reappear on the restoration of its force after the subsidence of the fever, a circumstance which we have often observed? I believe that in the present state of our knowledge, these questions cannot be answered with any degree of certainty.

In another case, with murmur loudest between the shoulders, there was still greater probability that the sign was inorganic.

A girl aged 12, was admitted into the Meath Hospital, in measles. The eruption was papular. She had been ill for a week, and the eruption was of two days' standing. The heart's action was excited, with a distinct systolic murmur, loudest at the apex. It was feebly heard at the upper part of the sternum, but along the left side of the spine, down to the loins, the murmur had a great degree of loudness. The pulse was 100, regular, strong, and full.

This child had been in hospital, twelve months before for an attack of chorea. The measles went through the usual course favourably, and the last report of the murmur, made on the thirteenth day, was, that it continued loud at the apex, feebler at the upper sternal region, but so loud along the spine that it could be heard all over the back.

The foregoing observations are not without practical importance, for in general, the danger which these patients run is not from the disease, but from misapprehension of its nature on the part of the practitioner. There exists in the minds of many men so strong a tendency to connect the ideas of the recent appearance of a murmur,—especially in connexion with fever, with inflammation,—that they are led to treat such cases as incipient carditis; and not only this, but also to fall into the error of proportioning the energy of their treatment to the importance of the organ engaged, and, in the excitement of the moment, to forget all the antecedents of the case, and, it may be, do irreparable mischief. All that is necessary in these cases is to direct a sufficiently tonic regimen, graduated according to the advancing strength of the patient, and with an eye to the period when the last symptoms of fever have disappeared. The preparations of iron appear proper when given moderately and in a continued course. But in truth, it is seldom necessary to adopt any treatment beyond that which common sense points out in the management of a patient convalescent from fever.

We have occasionally observed cases in which, before the attack of fever, anæmic murmur has existed. Such patients may go through the attack, and the murmur continue. Again, in persons of a chlorotic appearance, the murmur may become developed during or after the fever, and remain for an indefinite period. I have known this to occur even though the menstrual function had been re-established.

Before leaving the subject of anæmic or nervous murmur, I wish to notice a case which may give rise to conflicting opinions. It is the sudden development of a loud musical murmur, unattended with any circumstance in the history or state of the patient indicative of disease of the heart. I have known this to occur in middle-aged men, of fair complexion and delicate habit, and in whom there had been no rheumatic attack, and no complaint whatever of cardiac pain, palpitation, or of dyspnœa; the action of the heart and pulse perfectly regular, and the individual in the full enjoyment of all his powers of body and mind. In a case of this kind the patient cannot be persuaded that he is the subject of any disease whatever. There is good reason to believe that a fully developed musical murmur, nearly as loud as the mewing of a cat, may be produced in the course of one day. It is a single systolic murmur, and predominates at the left side, masking the first sound. In this way such individuals may continue for one, two, or three years, to all appearance in good health, and able to pursue the duties of a laborious profession. Gradually the signs of progressive disease of the heart show themselves. An attack of cardiac asthma may be produced from some indiscretion in exercise or regimen, and the pulse and action of the heart at length indicate that there is something organically wrong.

The total want of the usual antecedents of organic disease of the heart in these cases,—the absence of palpitation, pain, or irregularity of pulse, may mislead the practitioner, and make him refer the signs to some form of nervous murmur. The case illustrates what we have already said when speaking of the diagnosis of murmur with a quiescent heart. And it is important to be on our guard, and not to permit the want of the symptoms and history of heart disease to betray us into a diagnosis the error of which will be certainly shown in time.

It is not improbable that in cases of this kind there has been a silent process of disorganization going on in the tendinous cords of the mitral valves, and that, on the giving way of one of these cords, such an imperfection of the valves has followed as to cause murmur to be suddenly developed. I offer this merely as a suggestion, and as some explanation of the anomalous circumstance of the sudden appearance of signs which are commonly those of extensive and chronic disorganization.

The musical inorganic murmurs, with or without fremitus, appear to be less common in the heart than in the arteries.

It is laid down by authors, that inorganic murmurs generally are less common in the heart than in the arteries. This is probably true in the ordinary anæmic cases; but we must not forget that in fever, or its consequent states, an inorganic murmur in the heart is frequent.

Let us now glance at some additional phenomena often attendant on the nervous or the anæmic state. They may be enumerated as follow:—

1. Fremitus. It is generally co-existent with a rough or a musical murmur.
2. Doubling of the second sound of the heart.
3. Doubling of the first sound of the heart.
4. A sound of metallic ringing, attendant on the systole of the heart.
5. The contractions of the heart being attended by an abrupt sharp sound.

These phenomena, though often found in functional derangement, are not peculiar to it; they occur, though with less frequency, in many organic diseases. Indeed, cardiac fremitus is much more often seen attendant on important anatomical changes than on the anæmic or nervous state. Simply considered, we find it to be common in disease of the mitral and aortic valves, and it occurs in varicose aneurisms and imperfect closure of the foramen ovale. I do not know of any mere tactile or acoustic characters by which an inorganic can be distinguished from an organic fremitus. If inorganic murmurs be always systolic, the nervous or anæmic fremitus should follow the same law. It may be laid down as a general rule, that the bellows murmur and fremitus of anæmia are systolic in the heart; and when we reflect that in many, if not in all of these cases, the second sound remains unaffected, we may safely say, that inorganic fremitus is rarely if ever diastolic, double, or regurgitant. It may hence be affirmed, that when fremitus presents any of these characters, there is a presumption in favour of organic disease of the heart or the aorta.[1]

So great, indeed, is the rarity of inorganic fremitus of the heart, as compared with that in the arteries, that the existence of the purring thrill, under any form, in the heart, makes a strong presumption in favour of organic disease. In the arteries the case is different; for in them inorganic fremitus, even in a high degree, is by no means rare, requiring for its production a certain vehemence of the cardiac systole acting, it may be, on a column of impoverished blood. How far a local or general weakness of the arteries assists, we shall not now inquire.

In determining on the absence or presence of fremitus, it is sometimes necessary to use great delicacy in the manual examination; for there are degrees of fremitus so slight that we could not infer its existence from auscultation; in other words, there may be two patients, each presenting a bellows murmur of the same loudness and general character, and yet fremitus may exist in the one, and be absent in the other. Again, it may be present only at certain contractions of the heart. I have known it to completely elude observation when the heart was examined by simply placing the flat of the hand on the præcordial region, and yet to be distinctly perceptible when the tips of the fingers were lightly placed on the sixth intercostal space. In one case we found that pressure in this situation, when carried to a certain point, caused the fremitus completely to disappear, the bellows murmur, of course, continuing. Of this curious circumstance I can offer no explanation; but the observations were made repeatedly, and with great care. The patient was a boy, of about fourteen years of age, and of a delicate habit. He had been liable to palpitation of the heart from infancy, but he was able to run and perform the most active exercises. The entire of the first sound, at both sides of the heart, was replaced by a distinct, though soft, bellows murmur, loudest at the left side; the second sound was healthy; and at the upper sternal region the ordinary double sound of the heart could be heard. There was no arterial murmur, nor any appearance of cyanosis.

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[1] What has been now said is to be considered as more or less suggestive. Dr. Walshe has not met with regurgitant fremitus. I have lately seen a case of disease of the aortic valves with double murmur and single fremitus, the latter attending the second or regurgitant sound. This observation I made with great care. I have never satisfied myself of the diastolic fremitus which Skoda affirms he has found in disease of the mitral valves.

Finally, we may notice that species of vermicular sensation observed in cases of prolongation of the first sound in the anæmic state, consequent on typhoid fever. It scarcely deserves the name of fremitus; and a certain degree of care and delicacy in examination is necessary for its detection. It is a feebler sensation than that of ordinary fremitus; it is principally perceptible at the apex, and conveys the idea as if the muscles of the heart, in place of contracting, *per saltum*, presented successive and, as it were, vermicular motions.

I have never met with this phenomenon unassociated with the sound of muscular prolongation. The latter sign has been noticed by Dr. Walshe, who has suggested that it may be produced by slow contraction of the muscular fibre; but he has not, so far as I know, observed the accompanying tactile fremitus or vermiculation.

But the most important clinical point to be stated, in connexion with this sign and its associated sound, is, that they are in some way connected with a weakened state of the heart. I have only met them during the convalescence from essential diseases which had more or less of the typhoid type. It is curious that these phenomena, so common in the heart in typhoid fever, are yet so rare in true typhus. Is there a special nervous condition of the heart, or must this state be combined with some alteration of blood? In whatever way this question may be resolved, we find that the order of disappearance of the phenomena is, first, the vermicular sensation, and next, the sound of muscular prolongation; indeed, the latter may continue for a considerable period after convalescence, and when the heart seems to regain its natural force.

The next manifestation of deranged function of the heart has been already mentioned. It is the doubling, more or less perfect, of one of the sounds. I have never observed this sign to exist with both sounds at once, and have much more frequently found it in connexion with the second than with the first; in fact, doubling of the first sound is very rare; and itis rarely so well marked as in the case of the second sound; it is rather an attempt at doubling than a duplication of the systolic sound. On the other hand, when it occurs with the second sound, it is as if the latter were accurately divided into two parts, each of which has the clearness and defined character of the original sound. Its existence does not alter the rhythm of the heart's action.[1]

Doubling more or less complete, of either of the sounds, may be met with in the following cases:—

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[1] Dr. Walshe has some valuable observations of the reduplication of the sounds of the heart, of which I was not aware when the notice of its occurrence in some cases of carditis (see page 132) was printed. He explains the doubling of the second sound by a want of synchronism in the systole of the aorta and pulmonary artery. As to the doubling of the first sound, its explanation is more difficult; and we can only state generally, that it may, as Dr. Walshe suggests, arise from a want of consent between the ventricles. I have not observed the doubling of the second sound at the base, while it was single and free at the apex; nor again, the doubling of the first sound at one side, while it remained single at the other; but we may admit their occurrence on Dr. Walshe's authority. According to him, this doubling is not connected with any particular form of disease; it comes and goes in the course of a few beats of the heart; it sometimes disappears on change of position, and may be affected by the act of respiration. It occurs most commonly in hearts either healthy or but functionally disordered. It is commonly met with in slight organic disease, and is rare in cases of confirmed valvular lesion.—*Op. Cit.* p. 211.

1. *Nervous disturbance of the heart*, simply considered, or in connexion with anæmia. In this case the alteration much more commonly affects the second than the first sound.

2. *Endocarditis.*—Of this form I have already given three instances. Two of these patients were females. In one the doubling of the second sound followed the disappearance of the bellows murmur; and the double second sound was much louder than the first, though the action of the heart continued violent. In the second the disease was rheumatic: the first sound was that of distinct bellows murmur, while the second was replaced by two sharp sounds. The third, also, was arthritis, with cardiac complication, the physical signs being the same as in the last instance. During convalescence, the doubling of the second sound could only be distinguished in the horizontal position.

3. *Chronic bronchitis.*—In this affection, when occurring in very old persons, I have observed the sign, with considerable permanence, to engage the second sound. In this case murmur did not attend the first sound, and, with the exception of being weak, the heart appeared to be free from disease.

Lastly, we find, in a few cases of the permanently irregular and rapidly acting heart, that the first sound is either double or approaches to that character. This sign is less frequent where the irregularity is coupled with organic change than in cases in which, with permanent and extreme irregularity, we may still hope that organic disease has not supervened.

We cannot, therefore, admit with Dr. Walshe, that these reduplications are almost insignificant in the present state of knowledge; their occurrence in rheumatic endocarditis is of great importance, and they may, in all probability, be placed among those phenomena which, in a case of rheumatic fever, indicate a proclivity to carditis, if not the disease itself. Irregularity of action, increased force of the heart, depressed and slow action, doubling of either of the sounds, in short, any functional disturbance of the heart in a patient already labouring under rheumatic fever, should lead us not merely to expect the invasion of carditis, but to treat the heart as if it was already inflamed.

The last of the acoustic signs of the nervous heart to which we shall allude is the peculiar ringing sound sometimes attendant on the contraction of the ventricles. It is more common in cases where we meet great force, combined with regularity of action, than in the irregularly acting hearts. This is the metallic tinnitus of Laennec. Like other separate phenomena, it has probably a compound source; but that its principal cause is the energy of muscular contraction, with or without a great degree of tension of the auriculo-ventricular valves, there can be little doubt. It is common in the hysteric excitement of the heart, but occurs in many other cases.[1]

But we must not confound this metallic ringing of the muscular contraction with the peculiar character given to the heart's sounds from flatulent distention of the stomach and intestines. With this I have long been familiar, and have mentioned a case of double pleuro-pneumonia and pericarditis, in which all the physical signs of the chest received the metallic character in consequence of the distention of the abdomen. The crepitus and the pericardial friction signs were completely metallic. The use of a carminative draught removed this unusual modification.[2]

Dr. Corrigan has noticed the ringing sound in cases depending on gastric irritability attended with flatus. He describes it as a remarkably clear resonant sound, such as we might expect to be produced if the heart, when acting, lay upon a drum-head. He observes, that it is the first sound that presents this character most remarkably.[3] It is, however, probable, that where the ringing predominates in the systolic sound, it arises principally from the contraction of the heart itself.

Before describing some special instances of functional derangement of the heart, let us consider some points of practical importance which naturally arise from what has now been said.

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[1] Dr. Hope explains the metallic sound by the impinging of the apex against the rib during the systole, and observes, that he has never found the sound except in meager individuals. The decision of the question is not of any great practical importance, for, however produced, the ringing sound indicates an excited action of the heart. I have no doubt that valvular tension bears a great share in its production, for I have observed the ringing character to attend the second sound of the heart where it was altogether absent from the first. This occurred in a case of aneurism of the arch of the aorta in a young man. On the other hand, we have already seen that the *per saltum* action, even of the voluntary muscles, is capable of producing an extraordinary variety of sounds.

[2] See p. 43.

[3] On the Diagnosis and Treatment of some Functional Derangements of the Heart. Dublin Journal of Medical Science, First Series, vol. xix.

The differential diagnosis between nervous excitement of the heart and active hypertrophy is often a matter of great difficulty; and in many instances it becomes necessary that the patient should be seen on several occasions before we can arrive at a satisfactory conclusion. Confining ourselves to physical diagnosis, the following are the circumstances on which reliance is to be placed:—

1. The results of percussion.

2. The extent of the systolic impulse, and, as Dr. Corrigan has shown, the position of the apex.

3. The want of accordance between the apparent force of the heart and that of the pulse at the wrist.

Upon the first of these sources of diagnosis we need not dwell; and with respect to the second it has been already shown that, although in some cases of nervous palpitation an extended and apparently equable systolic impulse is met with, yet, by careful manipulation, we can determine that in this impulse there is a point of maximum intensity which corresponds to the natural situation of the apex.[1]

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[1] On this subject Dr. Corrigan, when describing a case of palpitation in a young growing person, observes, that "the pulse has become habitually quick, beating probably 120 or 130 in the minute; and on laying the hand over the region of the heart, the first impression made on the examiner is, that there is great hypertrophy of the left ventricle, as it is felt thumping over a large space, and apparently with great strength. A little further examination will, however, show that this is a deception arising from a combination of causes. The contraction of the ventricle is *quick*, and hence the impression of it gives a more palpable impression to the hand than a contraction of equal strength, but slower. The chest being generally narrowed or drawn in, the heart, as in females, lies with a greater extent of surface close to the ribs, and the hand of the observer feels necessarily the impulse over a larger space; and as this space increases, the hand laid over it will receive a corresponding increase of impulse, even though the strength of the organ be not increased, just as the hand laid over the large sac of an aneurism will, from increased extent of surface, receive a corresponding increase of impulse, even though the active force distending the sac is not greater than that exerted on a neighbouring artery, which gives an impulse immeasurably less to the perception of the observer. The deceptive sensation produced on the hand, when the heart gives a large extent of surface of impulse, is very likely, if its cause be not remembered, at once to make the practitioner suppose, in such a case as that described here, that he has to deal with a greatly enlarged and powerfully acting heart, when the organ is really the very opposite of this as to strength of muscular fibre.

"These observations are called for in relation to the class of cases I am now

The direction in which the apex points is of great importance. It is stated by Dr. Corrigan that he has never seen a case of hypertrophy with dilatation of the left ventricle, where post-mortem examination unequivocally proved the existence of the disease, that the impulse of the heart was not felt out of its normal situation. According to him, it cannot be otherwise; for, as the heart enlarges from base to apex, it makes room for itself, not by pushing out the cartilages of the ribs before it, but by turning its apex round more and more to the left side, according to the degree of enlargement, so that in cases of great enlargement the apex will be felt beating in a line dropped from the axilla. The distance to which the apex has travelled will be a measure of the degree of enlargement.

From these observations the rule may be deduced, that where there is no natural or acquired deformity, the beating of the apex, in its usual situation, will be an argument, in favour of nervous disease.[1] We, however, meet with cases of hypertrophy in which the direction of the apex remains unaltered.

Among the various signs of functional derangement of the heart there is none of greater importance than the want of pro-

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noticing; for if the idea be taken up, that auscultation will give the best information as to the muscular acting power of the heart, an error will, I am sure, very often be committed on this point, particularly when, as in such cases, the action of the heart is, as I have already observed, *quick*. In using the word *quick* here, it is to be understood in its accurate sense, not as synonymous with frequent, but as applied to the time occupied by *each* contraction of the ventricle. The heart and pulse may beat only 50 in the minute, and yet be quick, that is, each contraction may be so sudden as to occupy an exceedingly short space of time, and when either pulse or heart rises thus very suddenly under the finger, the suddenness of the impression gives a very deceptive sensation to the ear and finger. I know, from observing it frequently committed by the most intelligent clinical clerk, that such a mistake is easily made, and I have, therefore, thought it not amiss to notice it here. This error is always easily guarded against, by using two fingers to feel the pulse in doubtful cases, and ascertaining what degree of pressure of the finger nearer the heart is required to prevent the impulse from reaching the finger farther off. In this way it will often be found that a pulse, at first reckoned strong, will bear scarcely any pressure, although the arterial tube may be remarkably full."—*Op. Cit.*

[1] The acquired deformities may be of two kinds, either contraction of the chest from an empyema, or that prominence of the præcordial region described by Hope and Bouillaud in cases of great hypertrophy. It would be well to ascertain whether in the latter cases the direction of the apex is really changed.

portion as to force between the impulse at the side and the pulse at the wrist. And it is certain that the nature of this symptom is not yet understood. The difference between the action of a heart in which there is a quick, as it were, vivid contraction, and that of another in which, with greater power, there is less of suddenness, has been dwelt on by writers. And yet, while we admit the fact, it is difficult to understand why the apparent force of the heart, in the first case, is not equally perceived in the pulse. Is it that in these nervous or anæmic cases there is a want of that consent between the heart and arteries which seems to exist in inflammatory fever, or in certain instances of organic disease of the heart? The curious fact, that in some examples of nervous action of the heart, the arteries respond but partially, must here be noticed. If that peculiar disease of palpitation, with enlargement of the thyroid gland and eyeballs, be in its origin a functional lesion, as I believe it to be, we see in it this partial consent between arterial and cardiac action. Generally considered, the chief relation between the heart and arteries is as to time; yet in these cases the phenomenon of local correspondence in force occurs. The heart acts violently, so do the carotid and thyroid arteries, while the vessels of the extremities only show a feeble, though correspondingly rapid action. Is the symptom in question, then, of the want of correspondence between the force of the heart and the pulse to be explained rather by want of consent between the heart and arteries than by deficient power of the heart, though co-existing with a quick and forcible action?

We may now dwell on a few of the ordinary cases of deranged action of the heart which come before us in practice. Of these the following are examples:—

1. Increased action in young and growing persons.
2. Palpitation from derangement of the stomach.
3. Cardiac suffering from the abuse of tobacco.
4. Palpitation from the excessive use of tea.
5. Hysteric palpitation.
6. Rheumatic and gouty palpitations.

PALPITATION IN YOUNG PERSONS.

The examples commonly seen of this form occur in young men from sixteen to twenty years of age, who have outgrown their strength, and are of a delicate habit. Dr. Corrigan has noticed this form as connected with the process of growth; but what relation exists between this process and palpitation we cannot as yet declare. He observed a curious fact in one of these cases, that although the patient was unable to run, or use the ordinary exercises, without difficulty of breathing, yet that he did not suffer in the act of swimming.

The diagnosis must be founded on the previous history of the patient, especially in reference to the existence, even at a remote period, of the rheumatic state. We are then to observe the varying condition of the heart; its clear, sudden, and sometimes ringing contractions; the predominance of impulse at the apex, and in the usual situation; the absence of murmur, or, if it be present, its fugacious and uncertain character; the absence of signs of hepatic or pulmonary disease; and, generally, the want of proportion between the apparent force of the heart and that of the pulse at the wrist.

PALPITATION FROM DERANGEMENT OF THE STOMACH.

This is by far the most common case of functional derangement of the heart. Under a great variety of circumstances the heart's action may be merely increased in force, with or without pain. Its action is often perceived by the patient in unusual situations, such as the epigastrium, or high up in the thorax. Again, the disturbance may be paroxysmal, consisting of extremely rapid and fluttering motions, with intervals of perfectly normal action; or again, extremely violent and alarming symptoms arise, in which the paroxysm is protracted for many days, and the heart's action so vehement and irregular, attended also by various forms of bellows murmur, as to render it impossible for the physician who only saw the patient pending the paroxysm, to pronounce against the existence of organic disease. I have already given two examples of this form, in both of which the symptoms disappeared after the action of an emetic.

In dealing with these cases it is difficult to draw the line between palpitations resulting from mere sympathy with the stomach, and those produced by certain poisonous ingesta, which act on the nervous system, such as tea, tobacco, alcoholic drinks, &c. A common case met with is that from the abuse of tobacco. This often occurs with young men of the better class who have recently entered the army, and who have smoked to excess, in addition to other irregularities. It is rarely seen among peasants, or in men who have passed the age of twenty-five. The chief symptom is a throbbing action of the heart, sensible and distressing to the patient. It prevents exertion, especially on foot, and is aggravated by lying on the left side. In physical signs these cases resemble other examples of nervous palpitation. Irregularity of action and murmur are not common.

There can be little doubt that tobacco influences the nerves of the heart. The practice of swallowing tobacco-juice has been resorted to by malingerers in the army for the purpose of producing violent and irregular palpitations. Hellebore has been used with the same intent, but I have not myself met with either of these cases.[1]

DISTURBANCE OF THE HEART, CAUSED BY THE USE OF TEA.

In ordinary cases there are no special characters by which this form of palpitation can be distinguished from other examples of nervous disturbance of the heart; but when the abuse of tea has been carried to a great extent, important symptoms may arise. I shall quote two cases from the memoir of Dr. Edward Percival on the abuse of green tea:—[2]

"A gentleman, intending to walk some distance along the coast of Devonshire, set out in the morning of a hot summer's day, having previously breakfasted on strong green tea, a beverage to which he was not unaccustomed. Having walked twelve miles, he refreshed himself with a repetition of the same meal. Resuming his journey, he walked nine miles further, without hurry or fatigue. The heat of the day indisposed him to dine, as usual, on animal food, and he, therefore, called a third time for green tea, and drank copiously of a strong infusion, eating at the same time only bread or biscuit. He retired early to bed, resolving to use a similar diet on the following day.

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[1] I once observed in a case in which large doses of sulphate of quinine had been administered, the sudden production of an extraordinary palpitation. The action of the heart was tumultuous, irregular, and, to the highest degree, vehement and distressing. A loud bellows murmur attended the systole; but I cannot say whether it was propagated into the arteries.

[2] Some Brief Notices of the Deleterious and the Medicinal Effects of Green Tea. By Edward Percival, M. B. Dublin Hospital Reports, vol. i. 1817. As it is now established, that the green and black teas are in reality the product of the same plant, a fact which was not ascertained when Dr. Percival wrote his memoir, we may expect that similar results would follow the abuse of the black as well as of the green tea.

"Soon after he lay down, he began to feel some unusual and distressing sensations about the præcordia, as if he were continually on the verge of fainting. But being much disposed to sleep, these sensations were for awhile disregarded, and he passed two hours in a kind of troubled slumber, waking at short intervals. His respiration became irregular and oppressed; and his heart sometimes palpitated, and at other times seemed motionless. At length he awoke suddenly and entirely, as from a struggle of incubus. He now experienced acute pain, as from spasm in the region of his heart; and in spite of all his efforts he felt as if he were continually falling into deliquium. His pulse was feeble, irregular, and intermitting in an extraordinary degree, and slight fits of apparent asphyxia recurred every five or six minutes. He had with difficulty roused his servant at the inn where he lay, and procured (from an invalid companion of his journey) two opium pills, consisting of one grain each, and a small quantity of cold brandy and water. Deriving some temporary relief from these remedies, he again composed himself to sleep; but after an hour's slumber, almost distressing as that which he had before endured, he awoke in great agitation, gasping for breath, and bedewed with a chilly moisture. Another pill of opium was procured, and a glass of hot brandy and water, of greater strength than the former. From these he soon derived the wished-for relief, and at length fell into a sound and natural sleep, from which he awoke at his usual hour in the morning in perfect health. It deserves remark, that although perfectly unaccustomed to the use of opium or brandy, in any degree of dilution, yet he experienced neither thirst, headach, nor any other uneasy symptom from the remedies he had used in the preceding night. The bane and the antidote seemed mutually to cancel each other's noxious qualities. This gentleman has frequently used green tea, even strongly infused, since this occurrence, though never in the excess above described; and as he derives refreshment only, without inconvenience, from the beverage, it is reasonable to conclude that no peculiarity nor idiosyncrasy in his constitution occasioned the symptoms above detailed."

"A case analogous to this has been obligingly furnished me by Dr. Harvey, whose communication I beg leave to subjoin:—'Upwards of thirty years ago Dr. ——— called upon me in the middle of the day in the summer season. I happened to answer the door myself, as all my domestics were out looking at some public spectacle. He appeared to me to be actuated by great terror; and upon my asking him what was the matter, he said, "I have called upon you to request you would let me in, and allow me to die in your house." When he sat down I examined his pulse, which was scarcely discernible, and extremely irregular. He said he had called at the house of Dr. Hutcheson, and afterwards at Dr. Purcell's, but finding neither at home, he came to mine, where he entreated I would allow him to expire, which event he was sure was inevitable. I cannot say, at this distance of time, what the circumstance was which made me ask him if he had been drinking strong green tea. He immediately replied, that he had drank a great deal of strong green tea during the whole of the preceding night, as he sat up with an uncle of his who was to set off extremely early in a stage coach. I gave him a large glassful of cherry brandy, and put him to bed. He slept for a couple of hours, and awoke quite relieved from all his disagreeable feelings.'"

The following case occurred within my own observation:—A gentleman, of middle age, spare habit, and great intellectual attainments, had accustomed himself to pass a considerable portion of the night in literary and scientific labours. He was in the habit of making a large quantity of strong tea in the commencement of the evening, which he continued to drink during the night, without any thought or apprehension of its deleterious effects. At length the following condition was produced:—He became subject to paroxysms of quick and vehement action of the heart, which, however, was neither irregular nor intermitting; this was accompanied by intense præcordial distress and oppression, with a painful sense of impending death. The respiration was hurried and laborious, and these attacks recurred with such frequency and severity that this gentleman became convinced that he laboured under extensive disease of the heart and, probably, of the aorta. His spirits were depressed, and he had no expectation but that he should die in one of these dreadful paroxysms. The duration of the fit was uncertain; in the intervals there were no symptoms of any affection of the heart, and its action and sounds were perfectly natural.

But on the nervous system the effects of the poison were not thus remitting. His mind was unaffected except from the natural apprehension of death; but the following condition, of which I have never seen any other instance, had become permanent. He experienced the greatest difficulty in walking on level ground, so that in going through the city he was obliged to hold on by the rails which skirted the flagway. It was not that he suffered from giddiness in walking on a flat surface, but that he had an irresistible feeling that he should slip and fall, as a man might have in walking on smooth and polished ice. On one occasion, desiring to call on a friend who lived but a short distance from him, the apprehension of falling, produced by the sight of a broad and level flagway on which he had to walk, was so great that, after much deliberation and vain efforts to overcome the apprehension, and as time was pressing, he actually proceeded to his friend's house on all-fours. On the other hand, he never experienced the slightest difficulty in locomotion where the ground was rough or uneven. He could run up or down the most rugged mountain; could walk the deck of a yacht during a gale of wind with perfect facility, or on a level road, provided the surface was covered with rough shingle. This gentleman was not subject to imaginary diseases. His frame was strong; he had circumnavigated the globe, and borne the hardships of travel without injury.

On the occasion of my being consulted, the similarity of the cardiac symptoms with those recorded by Dr. Percival induced me at once to ask the patient if he had been indulging in strong tea. I showed him the cases in question, and having made a careful examination, was enabled to assure him that his symptoms did not proceed from an organic cause. He was advised to abstain from tea. The aromatic iron mixture was prescribed, and he was recommended to take a short sea voyage. The cardiac symptoms soon disappeared; but the apprehension of walking on a smooth surface, or, rather, the preference for any uneven path, remained with him for a considerable time.

The influence of an infusion of tea, especially green tea, in restoring regularity to a pulse habitually irregular, is noticed by Dr. Percival. The fact has been observed by others. I have no experience as to this point; but have more than once succeeded in producing refreshing sleep, by the use of green tea, in persons of a nervous habit, when opium and other narcotics had failed; and it is not impossible that this remedy might induce sleep in fever, when there is an excited heart and contracted pupil,[1]—a case generally unfitted for the use of opium.

DERANGEMENTS OF THE HEART'S ACTION FROM HYSTERIA.

A great number and variety of cases are to be grouped under this head. In a practical point of view, however, we may classify them as follows:—

1. Diminished action of the heart, as seen in the hysterical syncope or hysterical trance.
2. Fluttering and irregular palpitation, without apparent increase of the force of the heart.
3. Inordinate pulsation of the heart, in which its action sometimes attains to an extraordinary degree of vehemence, the force of the heart probably surpassing that which occurs in any case of organic or inflammatory disease.

I have seen some remarkable instances of hysterical syncope and trance; but not having made any observation as to the physical signs of the heart in these instances, I can offer nothing of novelty on the subject. In the second series there is little to distinguish the signs from those of ordinary nervous palpitation, and the diagnosis is to be drawn from the general history of the patient, the absence of positive signs of organic disease, and the previous or subsequent occurrence of hysteric disorder in other organs, attended by its characteristic anomalies and peculiarities.

The diagnosis is generally more difficult in those mitigated cases in which, under the influence of a marked hysterical state, the heart becomes more or less disturbed, than in those singular and often protracted examples in which the various functions are successively engaged, but in an isolated manner. Thus, where palpitation arises in a patient who has already been affected by hysterical mania, with hysterical dyspnœa or cough, with paralysis, convulsions, and so on, the nature of the affection of the heart can be often determined without difficulty.

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[1] Dr. Percival suggests the use of strong green tea in cases of poisoning by opium, especially where coffee has failed.—*Op. Cit.* p. 225.

It is in aggravated cases of hysteric excitement of the heart that we meet with the greatest amount of palpitation of which the organ seems capable. In some instances the force is so great as to agitate the entire trunk, and the bed on which the patient is lying. The sounds of the heart are often audible at a distance of some feet, and nothing can exceed the vehemence of its action under the stethoscope. Indeed, it appears strange that the valves or tendinous cords do not give way. Sometimes a corresponding action is seen in the carotids and the aorta, attended with murmur.

It is hardly necessary to state, that the sources of diagnosis are twofold:—First, the previous history and character of the co-existing symptoms; and next, the want of accordance between the physical signs as to duration and character, with those of the ordinary acute diseases of the heart.

A form of hysterical palpitation is met with in females consequent on the natural cessation of the uterine functions. It may continue for a considerable period of time, and is more liable to be excited by mental impressions than by bodily exertion. I have known the liability to paroxysms of the disease to continue for more than two years. The palpitations are described as fits of fluttering, with fulness in the chest and neck, attended with great anxiety and depression of spirits. In the intervals, the action of the heart and arteries is perfectly natural. In one case the cessation of the menses, which before had been very abundant and regular, took place suddenly in a lady, then aged 50, who had always been healthy, and not subject to hysteria.

RHEUMATIC AND GOUTY PALPITATIONS.

The occurrence of disturbed action of the heart in a patient of a rheumatic or gouty diathesis should awaken our closest attention. For although it is not to be denied that, under the disturbing influences of either rheumatism or gout,—palpitation, irregularity, or pain, may only indicate functional derangement,—yet we must look with apprehension upon them, if not with reference to immediate consequences, at least with regard to the future condition of the heart. With respect to rheumatic palpitation, as indicated by Bouillaud,[1] it may be laid down, that its occurrence is of less importance when it is unconnected with fever. Upon this everything depends; for, as we have before stated, disturbance of the heart's action, even though without any physical sign of inflammation, when arising in the course of rheumatic fever, is to be considered as showing the proclivity to, if not the existence of, carditis.[2]

The frequent immunity of the heart from valvular disease in apyrexial rheumatic disease, even though affecting the joints, has been already noticed. It is true, that in some cases of chronic rheumatic arthritis, we find that signs of disease of the heart are manifested towards the close of life. The aortic valves seem most often engaged, but many years may elapse before this complication is established. On the other hand, we observe that in patients who have at some former period suffered from an attack of rheumatic carditis, in which disease of the valves has been established, there is a liability to rheumatic neuralgia, affecting the centre of the chest or the right side, but most commonly the left side and the region of the heart. We have not found that any increase of disease of the heart followed these pains, though the patient, during their continuance, complained of præcordial distress. In one instance, which has been already alluded to, relief was obtained by taking violent exercise on horseback.

The following case might be called rheumatic neuralgia of the heart. It possesses some points of interest:—

A gentleman, aged 32, was affected with rheumatic fever three times during the last ten years. The articulations were always engaged; but there is no evidence that he ever had carditis. The second attack occurred five years since, and the third, three years. Each of these attacks lasted about a fortnight. It was after the second that he became subject to the symptoms in question. He has since had two kinds of suffering: one is a pain, referred to the upper sternal region, which he describes as like a spasm. It catches his breath, and the paroxysm continues for half a minute. This is brought on by any sudden bodily exertion. The other is excited by mental emotion in the waking state, or by dreams during sleep. It is more permanent, and is attended with a feeling of soreness, anguish, and agitation about the heart. It is remarkable that, during the last attack of rheumatic fever, there was a suspension or great remission of both these forms of suffering. In his ordinary state he is able to go through the duties of a laborious profession. The heart's action is regular, and there are no physical signs of disease in the heart or aorta.

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[1] Maladies du Cœur, p. 384.

[2] See Dr. Mayne's Researches. Dublin Journal of Medical Science, vol. vii.

It is to a disease of this kind that Bouillaud appears to allude when he speaks of palpitations which arise under the same influences as the ambulatory pains which are called rheumatic. He does not, however, give any case of this affection consequent on rheumatic fever.[1]

Excluding rheumatic fever, we may hold, that if, in a case of palpitation occurring in a rheumatic subject, it be hazardous to say that the heart is in a safe position, it becomes equally so in gouty palpitation.

This statement, however, requires some qualification. There appears to be a greater liability to the production of disease of the heart during the earlier attacks of rheumatism than of gout. Early attacks of rheumatic palpitation commonly eventuate in valvular disease, while the first or second access of gouty disturbance generally leaves the heart untouched, passing off, as is seen in dyspeptic palpitation. On the other hand, if we compare two patients who have long been subject, one to rheumatism, and the other to gout, there is a greater probability that any new disturbance of the heart indicates the commencement of organic disease in the gouty than in the rheumatic patient. Indeed, most cases of the so-called gouty palpitations are examples of a confirmed disease, which is an enfeebled and dilated state of the heart, combined with atheroma in the aorta.

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[1] Although this condition is noticed by Bouillaud under the general head of nervous affections, he does not specify the occurrence of pain. And probably from his adherence to the doctrine of the inflammatory origin of so many diseases, he expresses himself doubtfully as to the state of the cardiac nerves. The following are his words:—

"Il est des palpitations d'une autre espèce que les auteurs paraissent avoir aussi un peu trop négligées: je veux parler de celles qui apparaissent sous les mêmes influences que ces douleurs vagues ou *ambulantes*, que l'on connàit sous le nom de *rhumatismales*. Les palpitations en quelque sorte *rhumatismales* co-existent assez souvent avec une douleur dans la région précordiale, s'irradiant ou non vers le membre supérieur gauche. Les palpitations dont il s'agit coïncident parfois avec des intermittences du pouls, et causent habituellement une inquiétude extraordinaire aux sujets qui en sont tourmentés, bien que, sous tous les autres rapports, ils offrent pour la plupart les signes de la plus florissante santé."

"Il ne faut pas confondre ces palpitations avec celles qui ont lieu dans la péricardite ou l'endocardite rhumatismales: pas plus qu'il ne faut prendre une simple pleurodynie pour une pleurésie. Toutefois, je n'oserais affirmer que les palpitations qui accompagnent les affections rhumatismales des nerfs du cœur ne consistent pas, quelquefois du moins, en des lésions vraiment irritatives, mais mobiles, de ces nerfs; et si telle est, en effet, leur origine, elles ne sont pas alors une pure lésion d'innervation."—*Traité Clinique des Maladies du Cœur*, 1836, p. 334.

A mild form of palpitation may be observed in young men preceding a first or second attack of gout. The paroxysm often occurs at night, and the sensations are as if the heart was tumbling over, or rolling on itself. There is but little suffering or increased action, but in some cases a dull pain, or feeling of soreness, precedes or attends the paroxysm. On the following morning the patient, on getting out of bed, finds that the ball of one foot is swelled; the cardiac symptoms subside, and the whole paroxysm is mild, and of short duration. I do not know an instance in which such an attack was followed by cardiac disease. But when there have been repeated attacks of gout, especially in advanced life, the practitioner cannot be too cautious in attributing disturbance of the heart to gout, independent of anatomical change. Dr. Latham has insisted on this point.[1] With respect to those rapidly fatal cases which have got the name of gout in the heart, I have already expressed the opinion that they are examples of weakened heart which has long co-existed with the gouty state, and that at the termination of a protracted, severe, and, it may be, febrile, fit of the gout, the patient dies in a paroxysm of cardiac suffering, to which he has been rendered liable in consequence of erroneous antiphlogistic treatment. He does not die of gout in the heart, but he dies of the bad treatment of gout; and his death is caused by failure of the heart, the weakened state of which is so often overlooked.

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[1] See his Observations on Angina Pectoris, *Op. cit.* vol. ii. p. 416.

RECAPITULATION.

1. That angina pectoris, when occurring in middle or advanced age, indicates a weakened rather than a spastic state of the heart.

2. That it is most often met with in persons predisposed to fatty degeneration of the heart.

3. That although frequently co-existing with various forms of organic disease, the condition to which it is most intimately allied seems to be a weakened or degenerated state of the muscular fibre itself.

4. That obstruction of the coronary arteries must be considered as but a remote cause of angina.

5. That in a case of apparent neuralgia of the heart, the absence of physical signs of disease must not render us too confident as to the safety of the patient.

6. That the irregular and fluttering action of the heart, as seen in combination with certain enlargements of the liver, may exist or recur for many years, and yet suddenly subside, leaving the heart to all appearances in a perfectly healthy condition.

7. That in cases of this combination, it is often difficult or impossible to mark the period when the functional derangement of the heart becomes really complicated with organic change.

8. That another instance of the re-production of the normal state of the heart's action, after a long-continued disturbance, is to be seen in some cases of the pulsating thyroid gland and enlarged eye-balls. In these cases, too, organic disease of the heart may supervene; yet this change is not marked by any distinctive signs between organic and functional disease.

9. That inorganic murmur may arise, either with a regular or an irregular action of the heart.

10. That the diagnosis is less difficult in the first than in the second case.

11. That there is no certain acoustic character by which we can distinguish an inorganic from an organic murmur.

12. That the occurrence of murmur, with a tranquil heart, is more often met with in organic than in functional disease.

13. That inorganic murmurs, though systolic, are not always confined to the aortic orifice.

14. That in cases where the murmur is propagated into the aorta, the absence of regurgitant murmur, or, in other words, the clearness of the second sound, is an important diagnostic sign in favour of functional disease; for the only other case in which this combination has occurred to us is that of fatty disease of the heart, generally occurring in men of advanced age, in whom a certain amount of disease of the aortic valves is present.

15. That the occurrence of the anæmic state, in a case of mitral disease, appears sufficient to produce the symptoms of organic disease of the heart, and that these latter may subside on the removal of the anæmia, although the organic affection continues.

16. That an inorganic murmur may be developed in essential fever; but that it is much more common in the non-maculated than the maculated forms.

17. That it is more commonly observed in the convalescence, or intermissions, than during the fever itself.

18. That its point of greatest intensity is generally midway between the left nipple and the sternum.

19. That it frequently disappears or becomes diminished in the erect position.

20. That in connexion with some forms of inorganic murmur we observe the prolonged muscular sound. This is always systolic, and, in some cases, passes into true murmur. It may occur in various essential diseases.

21. That inorganic fremitus is much more common in the arteries than in the heart.

22. That the vermicular sensation, attendant on the prolongation of the first sound, may be described as a special form of inorganic fremitus of the heart.

23. That doubling of one of the sounds, though commonly a sign of merely nervous disturbance, is occasionally met with in connexion with inflammatory diseases of the heart.

24. That distinct doubling of the second sound is much more common than that of the first.

25. That the ringing, metallic sound of the heart generally indicates great vivacity and energy of contraction; but that in certain cases a metallic character is given to the heart's sounds in consequence of flatulent distention of the stomach.

26. That in the diagnosis between functional and organic derangements of the heart, we are to place our chief reliance on percussion, the extent of the impulse, the direction of the apex of the heart, and the relation between the force of the heart and that of the radial pulse.

27. That in certain cases of probably nervous disturbance, with increased force of the heart, a partial correspondence between the cardiac and arterial pulsation may be seen.

28. That as this correspondence is commonly found in the carotids, it is requisite that comparison should be made between the heart and the radial arteries.

APPENDIX TO THE PRECEDING CHAPTER.

Angina Pectoris.—Dr. Forbes, in his history of this affection (see the Cyclopædia of Practical Medicine, vol. i.,) and also the note to his translation of Laennec's chapter on Angina, has divided the cases into those of the functional and organic angina pectoris. Under the latter head he includes two classes; one in which the organic disease of the heart exists apparently as the sole malady; the other where, with more or less of organic change of the heart, there is some general disturbance of the system, so that the preventive or alleviating measures must be directed much to the latter state. Out of 45 cases in which dissection was performed, his statistical Table gives 39 in which organic disease of the heart or great vessels existed. In 4 there was "no organic disease except obesity;" and in two the liver was engaged. With respect to age, the Table gives out of 84 cases only 12 under fifty. In the Table of sex, the results were, that out of 86 cases not less than 80 were in men.

The cases of functional angina are divided by him into those without any organic lesion whatever, or even, as we infer from the text, without any complication with other disorders. He merely admits the probable occurrence of such a class of cases. But when conjoined with some other disorder, functional angina is by no means rare. This is his "*complex or sympathetic functional angina,*" under which he comprehends all the cases of nervous angina, complicated with other diseases, whether the organs of circulation are perfectly sound and well proportioned, or only deviating in a slight degree from the state of integrity. According to Dr. Forbes, a considerable proportion of the cases met with in practice, especially those which present great severity in the paroxysm, may be classed under this head. But this opinion, while received with respect, must be adopted with caution, especially when, in connexion with the pathological facts detailed in the text, the age, sex, and habit of persons most liable to angina are considered.

Dr. Parry refers to the account given by Seneca of his own case in the fifty-fourth epistle to Lucilius. It had been suggested to him that the symptoms there described were those of angina pectoris. I shall give the passage in full:—

"Longum mihi commeatum dederat mala valetudo: repente me invasit. Quo genere? inquis: prorsus merito interrogas: adeo nullum mihi ignotum est. Uni tamen morbo quasi assignatus sum: quem quare Græco nomine appellem, nescio. Satis enim aptè dici suspirium potest. Brevis autem valde, et procellæ similis, impetus est: intra horam fere desinit. Quis enim diu expirat? Omnia corporis aut incommoda, aut pericula, per me transierunt: nullum mihi videtur molestius. Quid ni? aliud enim quicquid est ægrotare: at hoc est animam agere. Itaque medici hanc meditationem mortis vocant. Faciet aliquando spiritus ille, quod sæpe conatus est. Hilarem me putas hæc tibi scribere, quia effugi? si hoc fine quasi bona valetudine delector, tam ridicule facio quam ille quisquis se vicisse putat cum vadimonium distulit. Ego vero et in ipsà suffocatione non desii cogitationibus lætis ac fortibus acquiescere. Quid hoc est? inquam, tam sæpe mors experitur me? faciat, at ego illam diu expertus sum. Quando, inquis? antequam nascerer. Mors est, non esse id quod antea fuit: sed quale sit jam scio: hoc erit post me, quod ante me fuit. Si quid in hac re tormenti est, necesse est, et fuisse antequam prodiremus in lucem: atqui nullam sensimus tunc vexationem . . . . His et hujusmodi exhortationibus tacitis (nam verbis locus non erat) alloqui me non desii: deinde paullatim suspirium illud quod esse jam anhelitus cœperat, intervalla majora fecit et retardatum est ac remansit. Nec adhuc, quamvis desierit ex naturâ fluit spiritus.

Sentio hæsitationem quandam ejus et moram. Quomodo volet dummodo non ex animo suspirem,"[1] &c.

We must agree with Dr. Parry in the opinion that the symptoms here detailed are not those of angina pectoris. It is remarkable that the occurrence of pain is not alluded to. But their similarity to that abnormal respiration, already described as attendant on the fatty heart, is too obvious to be overlooked. For in this affection we see that special form of dyspnœa which may be described as a paroxysm of sighing. Seneca's words, "Satis mihi apte dici suspirium potest;" and again, "Brevis autem valde, et procellæ similis, impetus est," are singularly expressive of a severe case of the cardiac sighing observed in persons labouring under fatty heart, for which, when the highest point of suspirious breathing has been reached, we can have no better comparison than that of a storm. And the words, "Deinde paullatim suspirium illud quod esse jam anhelitus cœperat, intervalla majora fecit et retardatum est ac remansit," well express the gradual ascent from what we may term the apnœal period to the extreme point of the paroxysm, and its subsequent decline.

Occurrence of bellows murmur at the upper portion of the left side of the chest.—Dr. Latham and Dr. Hughes have noticed this phenomenon as occurring in certain cases of tubercular phthisis.

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[1] In order to assist the reader in coming to a proper conclusion as to the nature of the case of Seneca, it is important to note that, according to Lipsius, the Epistles to Lucilius were written during the two years preceding that in which he was put to death. This would make his age at the time of writing the Epistles sixty-one or sixty-two. In several places he speaks of himself as being infirm and old. And Tacitus informs us, that at the time of his death he was feeble and exhausted,—"Corpus parvo victu tenuatum." From an early age the health of Seneca appears to have been delicate. Speaking of his aunt, Seneca says: "Illius pio maternoque nutricio per longum tempus ægre convalui." (*Consol. ad Helv.* cap. xvi.) This was written after he had been brought to Rome from his native place, Corduba. In one of the Epistles to Lucilius he speaks of having suffered from catarrhal fluxes,—"distillationes,"—to an extreme degree; and a friend suggests that these circumstances may account for the story told of him by Dio:—"Cum causam quandam in Senatu egregie et feliciter Seneca egisset, livore suffusum illum principem, qui solitus videri eloquens volebat, cogitasse de Senecâ tollendo: et fecisset, nisi una e concubinis admonuisset. Frustra mortem parari jam morituro: tabe quippe laborare." Influenced by this statement, Caligula spared the life of the young orator, verifying the remark which Seneca may have made with referenee to his own case, "Multorum mortem distulit morbus, et saluti illis fuit videri perire."

I apprehend that they both speak of the murmur which I described in my work on Diseases of the Chest, as among the signs of tubercle connected with the circulating system. The seat of this murmur appeared to me to be the subclavian artery; and the curious fact was noticed of its temporary subsidence after hæmoptysis, or the application of leeches. It is described by Dr. Latham as met with in persons who were undeniably consumptive, or partly suspected of being so, and to occur in a space bounded above by the upper edge of the second, and below by the lower edge of the third costal cartilage, extending along each rib for the distance of an inch. It is, according to him, a gentle bellows murmur coincident with the systole of the heart, and not heard in the præcordial region, the aorta, or carotids.

Dr. Hughes observes that this murmur may arise from many other diseases besides tubercle; namely, accumulations of any kind in the lung; thickened pleura, or mediastinal tumour; and he considers that its cause is mechanical pressure upon the aorta or pulmonary artery. It is, according to him, heard only during inspiration, so that it coincides, on the one hand, with the ventricular systole, and on the other with the act of inspiration. It is, doubtless, more distinct during inspiration, but to say that it was only audible at that time would be incorrect. Although it occurs in other diseases besides phthisis, we must admit that it is more common in that disease than in other thoracic affections. It is difficult to subscribe to the opinion of Dr. Hughes as to its mechanical origin; for we often find it in the earlier periods of phthisis; and, as has been now just stated, it may appear and disappear within short spaces of time. We must consider it, at all events, as among the collateral signs of phthisis. I have generally met with it at the early or the middle periods of the case, and have not only observed its temporary subsidence after an attack of hæmoptysis, but also its manifest increase immediately previous to such an accident.

This form of murmur is also met with in cases of acute pleurisy of the left side, especially when attended with excitement of the heart. It is distinct from any modification of the friction sound, and consists in a systolic murmur, often broken into two parts, and sometimes intense. This murmur is most evident during inspiration, but it continues in expiration, and even when the patient holds his breath. It may disappear wholly within thirty-six hours. We cannot, then, admit with Dr. Hughes, that this curious murmur is only heard during inspiration.[1]

Doubling of the Second Sound.—The following observation is worthy of being recorded as an addition to our knowledge of this subject:—A clergyman, aged 28, in consequence of over-exertion, showed symptoms of phthisis about three and a half years since, but the disease, in consequence of judicious treatment and the effect of a change of climate, which had been recommended by Dr. Williams, did not run its ordinary course, but passed into that indolent and chronic condition in which disorganization advances slowly, the constitutional symptoms being modified or suspended, and the physical signs rather those of dilated tubes than of ulcerous cavities. I saw this gentleman for the first time on his return from Australia. The whole of the upper and anterior portion of the left lung was comparatively, if not absolutely dull, with imperfect vesicular murmur, and a large mucous râle. During rest, the pulse was 80, and regular, and the heart's action tranquil. There was no murmur, but the second sound, as observed at the base of the heart, was most distinctly doubled. This character was evident over the whole antero-superior portion of the left side, but when the mesian line was crossed, it completely disappeared, leaving the sounds of the heart natural. At the nipple, too, the doubling of the second sound was extremely rare and indistinct. Indeed, when the heart was excited, no doubling whatever could be observed in this situation; it was only to be found during rest, when it occurred at intervals, and in a most indistinct manner.

Dr. Walshe[2] inquires how this curious phenomenon can be explicable on the sigmoid theory of the second sound. He states, that the sound may be single at the base, and double at the apex. This I have not observed. Every one must agree with Dr. Walshe as to the importance of these reduplications with reference to the doctrine of the heart's sounds.

Anæmic Murmurs of the Heart.—Dr. Hughes observes, that the anæmic murmurs are never heard at the bottom of the sternum. This statement is, perhaps, too decided. I am strongly under the impression that not only the prolongation of the first sound, but also the murmur, in fever, may be occasionally found under the lowest portion of the sternum. The same author observes, that the anæmic murmur, according to his experience, is never observed with a soft, slow pulse. Here, again, the statement has to be corrected by referring to the phenomena of fever; for the murmurs and prolongation of the first sound were found by us most frequent in the convalescence, and when the pulse had lost its force and rapidity.

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[1] Guy's Hospital Reports, vol. vii. p. 171.

[2] Op. cit. p. 211.

In discussing the differential diagnosis between organic and inorganic murmurs, Dr. Hughes observes, that where obstruction or regurgitation exists, the strength of the radial pulse, as compared with the impulse of the heart, will necessarily be in some measure diminished, and its character in some degree changed. "Though the impulse of the heart," he says, "be sharp and powerful, the pulse at the wrist may be, and often is, small and feeble. But in anæmia, without persistent and actual physical obstructions, the character of the pulse at the wrist coincides pretty exactly with that of the heart's impulse. It is hence obvious, that as a want of due relation between the one and the other is indicative of the organic disease, so the existence of that due relation is characteristic of the functional disorder."[1]

It has been already shown that, in certain cases of functional disorder, a vehement action of the heart may exist, while the pulse at the wrist does not correspond except as to time; and again, that we observe in fever the curious condition of a forcibly acting heart, with a pulse of the last degree of feebleness, or even extinct. The exact relation between the inorganic phenomena of the heart in fever, to those which occur in ordinary cases of anæmic or nervous disturbance, is not yet fully determined; but it is certain that we observe in this disease the co-existence, on the one hand, of a feeble heart with a pulse of ordinary strength, and on the other, of forcible action of the heart, with singular feebleness, or even cessation, of the pulse.

Murmur occurring in the last periods of life.—This has been noticed by Dr. Latham. "In these cases," he says, "the act of dying was slow and lingering, and although the murmur was declared a few days before death, it did not arise until the dissolution might be said to have already begun."[1] The conjecture of Dr. Latham, that this murmur may be produced by coagulation of the blood, is probably correct. I have given a case of the development of murmur shortly before death in cholera, in which a long coagulum extended from the cavity of the ventricle into the aorta; other causes, however, may produce it. Thus, in a case communicated to Dr. Latham, a bellows murmur became developed for a short time before death in a patient who had an affection of the brain; both sounds were attended by a murmur; and with the first there was a barking or yelping sound, like the voice of young puppies. In this case there was no coagulum, but the left ventricle was in a state of extreme contraction (concentric hypertrophy;) the valves were healthy. This case may have been one of endocarditis, developed during the last periods of existence.

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[1] Op. cit. p. 270.

Influence of position of the body on the rate and action of the Heart.—We may derive some assistance in the diagnosis of functional and organic disease of the heart by comparing the rates of the pulse in the horizontal and the erect position. The researches of Dr. Graves have shown that the influence of position on the pulse is less observable in cases of hypertrophy of the heart than in those where the organ is free from disease. He observes, that in healthy persons the pulse is more frequent in the erect than in the horizontal position by from six to fifteen beats in the minute. If the pulse is but 60, the difference is generally not more than six or eight, and this difference increases with the frequency of the pulse at the time of the experiment,—thus, if it has been raised from 60 to 100, by moderate exercise, it is not unusual to find the difference 20 to 30. The pulse, too, according to him, is stronger in the horizontal than in the erect position, consequently its maximum of strength and minimum of frequency are attained together. And he applies this fact to the explanation of the relief produced by the horizontal position in syncope. This difference has been found also in many diseases, including fevers; but in six cases of hypertrophy with dilatation of the heart, Dr. Graves ascertained that no such difference was perceptible, although all these patients, at the time these experiments were made, were in a debilitated state, in which the changes produced by position are the most remarkable. In four cases the existence of hypertrophy with dilatation was proved by post-mortem examination. When the upright position is attained by the patient's own exertions, a slight acceleration was observed during a time not exceeding a quarter of a minute.[1]

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[1] Op. cit. vol. i. p. 57.

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[1] Dr. Thompson, in his work on Inflammation, remarks, that there is a considerable difference between the frequency of the pulse in the erect and horizontal position in health, and still more in disease. These observations are acknowledged by Dr. Graves, who further remarks, that "Authors who have written concerning the effects of digitalis on the organs of circulation, speak of the difference between the pulse as observed in different positions, as an inexplicable anomaly, and seem quite ignorant that a similar phenomenon occurs in a less degree in health, and in an equal degree in many diseases. The fact appears to be, that *digitalis*, besides a great and debilitating influence on the whole constitution, and particularly the nervous system, possesses a *peculiar* power of diminishing the frequency of the pulse; *but it is no anomaly* that, in persons under its influence, debilitated and nervous as they always are, when it is exhibited in doses sufficient to retard the pulse, there should be a great difference between the frequency of the pulse as examined in the horizontal, the sitting, and the erect postures.

"I need scarcely add, that I cannot advance even a plausible conjecture concerning the reason why a change of position should so affect the frequency of the pulse. It is singular enough, however, that Humboldt should have observed something similar in the hearts of frogs, cut out of the body, the great vessels being tied. In one of these experiments the heart being placed on a piece of glass horizontally, after twelve minutes its pulsations had sunk to 12 in a minute. It was now suspended perpendicularly, and after two minutes the number of pulsations rose to 20. Baer, in his work, "Ueber entwickelungs geschichte der Thiere," &c., has made the curious observation, that in hatching eggs artificially, the chick in ovo soon dies if the egg be so placed as to rest on either end. This circumstance, which he does not attempt to explain, suggests an obvious and beautiful explanation of the reason why eggs are not round but oval, as the latter shape effectually prevents them from assuming a position in the nest which would be fatal to the enclosed fœtus. Some ova, as, for instance, those of certain reptiles, are round; but I know of no bird whose eggs are not more or less oval. It would be interesting to investigate the cause of this phenomenon, as also to examine into the reasons of the remarkable difference which exists between the effects of position on the human fœtus in utero, and on the human adult. In the former the inverted or semi-inverted position of the body is the natural position: in the latter it is unsupportable for any length of time."

The following are the conclusions arrived at by Dr. Graves upon the effect of change of posture on the frequency of pulse in other diseases and conditions:—

"1st. That the greatest difference occurs in patients labouring under fever, or in a debilitated state, in consequence of fever or any other cause. It may amount to 30, 40, or even 50, between the horizontal and erect postures.

"2ndly. That this difference decreases after the first quarter of an hour in most cases, but always remains considerable as long as the same position is observed.

"3rdly. That in persons not much debilitated, the difference is much less than that stated above, and often does not amount to more than 10.

"4thly. That when the patient lies down, the pulse rapidly falls to its former standard.

"5thly. That in some the increase in frequency is greater between the horizontal and sitting posture than between the latter and the erect; while in others the contrary takes place, so that, generally, the frequency in the sitting posture may be taken as a *mean*.

"6thly. In persons convalescent from fever or acute diseases, I find it is extremely useful to the physician to ascertain the comparative frequency of the pulse in the horizontal and in the erect position. The greater the difference, the greater is the debility of the patient, *and consequently the more guarded must his medical attendant be in allowing him to sit up for any length of time*, particularly if the pulse on his lying down does not resume its usual degree of frequency."[1]

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[1] On the Influence of Position upon the Heart and Pulse. By R. J. Graves, M. D., &c., Dublin Hospital Reports, vol. v. These observations are confirmatory of the remarks of Sir Astley Cooper (see his Lectures on Surgery,) relative to the increase of rapidity of the pulse in cases of concussion of the brain, when the patient is changed from the horizontal to the upright position.

CHAPTER XI.

ANEURISM OF THE THORACIC AORTA.

The local and proper signs of aneurism are the impulse or impulses,—the sounds, attendant on the pulsation, which may be double or single,—and lastly, the murmurs. These latter, however, are so often wanting, that we can scarcely consider them as proper signs of aneurism, at least of the thoracic aorta.

The production of the first two sets of signs, namely, the impulses and sounds of diastole and systole, seems to depend greatly on the localization of the disease. Hence they are best seen in sacculated true aneurisms, and in false aneurisms which are well defined and not of the greatest size. They may occur in the left sternal region, in the infra-clavicular spaces, at the upper portion of the sternum, the interscapular spaces, or in the right mammary region. I have seen a case of a true sacciform aneurism of the ascending aorta—the ampullar aneurism of Cruveilhier—presenting in the last-named situation. In the majority of cases these aneurisms are not attended with manifest external tumour, or other alteration of the surface, yet the pulsation can often be detected by the eye, and, if the patient be not too corpulent, the existence both of the cardiac and aneurismal pulsation,—the appearance, as it were, of two hearts beating in the chest, in different situations, is sufficiently plain. In other cases, the aneurismal beat is not so plainly visible, but here, as Dr. Greene has observed, if the eye be brought down to a level with the chest, looking across the thorax, we may perceive either a localized pulsation, or a diffused, but distinct throbbing in the upper sternal, or infra-clavicular regions. I am unable to say whether, in cases of double impulse, the eye can detect the second stroke so commonly observed in the heart, but it must be recollected that in the heart the seat of the second stroke is different from that of the first.

Dr. Hope, referring to the physical signs of aneurism generally, has stated that, although often closely resembling those of the heart, they may be distinguished by "*unequivocal criteria.*" This proposition cannot be admitted in its full sense. It is true that in many cases the impulses of an aneurism have a character different from that of the heart in the state of health or of active hypertrophy. This difference is more easily learned than described. It may be stated, that the aneurismal beat generally gives the idea of a forcible blow, having a force equal in all directions, while that of the heart conveys the sensation of a mobile but solid body, which, in many instances, at least, presents its greatest force at a particular point.[1]

There are no proper signs by which the pulsations of an aneurism within the chest can be distinguished from those of the heart,—none deserving the name of unequivocal. We commonly find that two sounds, singularly resembling those of the heart, are produced by the diastole and systole of the sac. In some cases the sounds of this apparently second heart have in every respect the character of those produced by the real heart. In others we may observe some shades of differences between the sounds emitted by these two centres of pulsation; but even here, if we take the aneurismal sounds alone, they may be often held to be exactly similar to those of a healthy heart. There is the same rhythm, the same comparatively dull and muffled character of the first sound, while the second is sharper. It commonly happens, too, that no trace of murmur can be discovered with the aneurismal sounds, and then the resemblance to the sounds of a healthy heart is complete. We may thus have two cases in which the aneurismal sounds are not distinguishable from those of the heart by unequivocal criteria.

I. A case in which the sounds both of the aneurism and of the heart have the same character in all respects.

II. A case where, in the particular individual, there may be some differences between the sounds of his heart and those of the aneurism, yet the latter not differing from the sounds of the heart as they are generally met with.

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[1] I have lately had occasion to observe an exception to this rule in a case of weakened heart. The pulsation of the ventricle had much of the aneurismal character, which is what we should expect if the tonic contractility of the ventricle was much injured. I have referred to this case under the head of Fatty Degeneration of the Heart.

The difference in this case may be, that the second sound of the aneurism is louder than that of the heart, and it sometimes has a ringing character, more perceptible when we apply the naked ear than by the stethoscope.

Hence, when we consider the general similarity of the two sets of sounds, and the frequent absence of murmur both in the heart and the aneurism, we must allow the diagnosis of an intra-thoracic aneurism to depend, in many cases, on the discerning of two centres of pulsation,—of two hearts, as it were, within the chest. It of course often happens that other acoustic signs will assist in the diagnosis.

In order to detect the pulsation it is sometimes necessary to make pressure with the flat of the hand on the anterior part of the chest, while the other hand is placed between the shoulders. If the aneurism be at all perceptible to the touch, it will often be detected at the end of expiration, though it becomes imperceptible during inspiration, and when the chest is fully dilated. Similar circumstances are to be observed in the case of a weakened heart.

There are two diseases, which, from the great amount of arterial throbbing attendant on them, lead to the suspicion of an aneurism, although no such disease exists. These are the common cases of permanently patent aortic valves, and gouty irritation of the aorta. The diagnosis in the first case presents no difficulty; the peculiar throbbing pulse perceptible over a large portion of the arterial system, the visible pulsation of the arteries, the increased action of the vessels of the neck, and the double bellows murmur in the ascending aorta, or the arch, all declare the nature of the disease.

Of the second case I have seen one remarkable example. It occurred a good many years ago, and presented so many of the physical signs of aneurism, and also such remarkable symptoms, that I was almost certain of the existence of a large aneurism of the aorta. The patient recovered, and lived for several years without any symptoms of aneurism whatever. It is probable that his disease arose from gouty aortitis. In addition to the existence of an intense throbbing, confined to the upper sternal region, this patient experienced the greatest distress in respiration whenever he assumed the horizontal position, so that for several weeks he had to remain sitting up. Not having any notes of this case, I am unable to say whether bellows murmur or double sounds were present, but the symptoms were such as to leave no doubt on my mind at the time that the patient had an aneurism of the aorta.

It may be laid down that in most cases where the action and force of the heart is normal, the occurrence of a diastolic pulsation of the upper sternal or the infra-clavicular regions, indicates aneurism, provided that there is no increased and visible pulsation of the carotids, nor the regurgitant murmur of permanent patency. Indeed, I know of no other disease competent to produce this group of circumstances unless it be cancerous tumour; but such a case, fortunately for practical diagnosis, is one of rare occurrence.

Signs from Percussion.—We are yet in want of an extended series of observations on this subject, and I have little to add to what has been already recorded. Laennec and Hope have merely indicated the occurrence of dulness in intra-thoracic tumours. Dr. Walshe, however, has discussed the point with more care, and holds, that percussion is valuable, not only in detecting even a small aneurism when in a certain situation, but also in enabling us to determine whether the aneurism contains a great quantity of coagula, or is filled almost wholly with fluid blood. According to this author, a dilatation of the vessel, of about two-thirds of its natural diameter, was observed by percussion. In this case there was thrill, and also increased impulse. The dilatation was at the right angle of the arch, where the vessel approaches the surface most closely. Dr. Walshe is of opinion that, although where a small sac is situated at the posterior portion of the vessel, it will be difficult of detection; yet that by careful percussion, an aneurism, not larger than a good-sized walnut, may be discovered if it be between the second right and left intercostal spaces.[1]

I can neither affirm nor deny the statement of Dr. Walshe,—that percussion will enable us to distinguish between an aneurism containing much fluid blood, and one in which coagula predominate; but I have seen cases in which it was impossible to detect an aneurism by percussion. Even where there were undoubted physical signs and symptoms of the affection, yet still no dulness on percussion whatever could be discovered. Inequality of respiration, dysphagia, and even copious hemorrhages, may occur under these circumstances. The absence, then, of localized dulness on percussion should not make us conclude against the existence of an intra-thoracic tumour.

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[1] Op. cit. p. 242, 549.

It must be added, that both the dulness on percussion, and the localized single or double pulsations, may disappear from their original situations without any cure of the aneurism, but solely from the tumour pointing in another direction. Phenomena of this kind are probably peculiar to cases of false aneurism.

SOUNDS OF ANEURISM.

These are to be considered under the heads of the proper or pulsative sounds, which, like those of a healthy heart, occur without any form of bellows murmur; and next, of the murmurs, which, in some cases, attend these sounds.

The common occurrence of a bellows murmur, in cases of arterial disease, not aneurismal, and in aneurisms of vessels of the extremities, have led to the too generally received notion of the value of murmur, or of the want of it, as indicative of the presence or the absence of aortic aneurism. But it is so commonly wanting in thoracic aneurism that I am disposed to consider its occurrence as exceptional. It appears to be an accidental phenomenon, evidently arising from mechanical conditions either of the sac, heart, or vessels, which are anything but constant.

We may divide cases of aneurism, with reference to the occurrence or non-occurrence of bellows murmur, into three classes:—

1. Cases in which it is absent, the signs being those of a single or double pulsative sound without any murmur whatever.

2. Cases in which a murmur, which is produced by the disease itself, is found to exist.

3. Cases in which a murmur is communicated from the heart. This arises when we have the combination of aneurism, whether true or false, with disease of the aortic valves.

In the second class we find the murmur confined to the locality of the aneurism, and the signs of enlargement of the left ventricle and of disease of the valves are wanting. In the third class, on the other hand, there commonly exist, in addition to the signs of aneurism, those of the condition of the heart which attend the permanently patent state of the aortic valves.

It will be seen that I differ from Dr. Hope with respect to the character of aneurismal sounds. He says: "The first aneurismal sound, coinciding with the pulse, is different from the first sound of the heart. It is a murmur varying, indeed, in its pitch, and softer or rougher according to the circumstances of each case, but still a murmur; and it is to this murmur that the loudness of the sound is attributable when it exceeds that of the ventricular systole."

But, in fact, there are two kinds of aneurismal sounds,—those attended by murmur, and those in which there is no murmur whatever. When aneurisms of the latter class have but a single sound, it has the closest resemblance to that of the ventricular systole; but when the sounds are double, they are so similar to those of the heart, that, were a good observer blindfolded, and the stethoscope placed for him over the seat of disease, he would find it difficult, if not impossible, to distinguish them from the ordinary sounds of an excited heart. This occurs both in the true and false aneurisms of the thoracic aorta, which may present these sounds without any murmur whatsoever.

These remarks do not apply to the case of abdominal aneurism, in which pulsation is commonly single, and, I believe, in most cases attended with some form of murmur. When murmur occurs, however, its characters are those so well laid down by Dr. Hope, who describes it as "a deep, hoarse tone, of short duration, with an abrupt commencement and termination, and often, but by no means invariably, louder than the most considerable murmurs of the heart."[1] The same author mentions "the varying character of the murmur at different portions of the tumour, and the lesser degree of hoarseness and loudness in abdominal aneurism."[2] Both of these observations we have often confirmed.

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[1] Op. cit. p. 416.

[2] Op. cit. p. 418.

In cases with a special or intrinsic murmur, we are still ignorant of the exact condition which determines its presence or absence; but there is less difficulty where the murmur is communicated from the heart. When disease of the aortic valves exists, the murmur is carried along the artery, and may be multiplied or increased in the aneurism. So far back as 1834 I observed that bellows murmurs were frequently absent in aneurism, and stated that we were in want of facts to explain their presence or absence in particular instances. The condition of the vessel at the distal or at the cardiac side of the aneurism appeared to me to explain the occurrence of murmur better than the state of the aneurism itself, just as the murmurs of the heart depend so commonly on the state of its orifices.[1]

Dr. Corrigan has since proposed an explanation by referring to the condition of the aortic orifice. If this is diseased, so as to give murmur, we have it also in the aneurism; but where the second sound of the heart is healthy, the aneurism beats without murmur. We may thus explain the murmur in the combination of aortic aneurism and disease of the aortic valves; yet murmur occurs in thoracic aneurisms where the valves are sound. In abdominal aneurisms it is almost constant; yet the combination of disease of the muscular structure of the heart or of its valves is very rare.

It has been supposed that the production of murmur arose from pressure exercised on the sac in the same way as it is developed in a large though healthy artery, when the compression is sufficient to diminish the caliber of the vessel, but not to obliterate the canal. Yet, on comparing thoracic with abdominal aneurisms, we find that murmur is much more common in the latter, although in this case there is less liability to pressure. In an example of aneurism of the arteria innominata, presently to be given, the tumour never produced dislocation of the clavicle, and so great was the pressure that a deep sulcus was produced in the line of the bone, and the trachea was pushed far to the opposite side. Yet no murmur ever existed in this case.

We are, then, still unable to explain the occurrence of murmur in aneurisms. Its presence or absence, however, is not a matter of great importance, at least in the case of thoracic aneurisms. But if a murmur, particularly the hoarse, abrupt murmur described by Dr. Hope, is met with as the single physical sign, or confined to a certain portion of the artery, there will be good reason to suspect aneurism.

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[1] Researches on the Diagnosis and Pathology of Aneurisms, Dublin Medical Journal, vol. v. p. 418.

I have seen two cases, both in females, where a loud and localized aortic murmur, not proceeding from the heart, existed, yet in which the disease was not aneurism. In one the murmur was heard in the lower portion of the dorsal region; it occupied a well-defined space of about two inches along the spine, and was exceedingly loud. The patient was a lady, of middle age, of a nervous habit and debilitated system. No thoracic or abdominal pulsation could be discovered by the hand, and the lower portions of the chest were clear on percussion; the heart was healthy, and the ordinary symptoms of aneurism wanting. The character of the murmur, too, was different from that of aneurism generally. It was a painfully loud and musical murmur. All this made against the diagnosis of aneurism, yet we could not but look on the case with great anxiety. This lady, however, recovered slowly under change of air and tonic medicines.

The second case was one of chlorotic anæmia, but its peculiarity was in the singular localization of the murmur. It was developed at the junction of the third rib with the sternum, on the right side, and attended with a distinct throb, and the vibratory sensation of aneurismal varix.[1] There was no murmur in the heart, nor in the vessels of the neck. This patient, a girl of about 18 years of age, recovered perfectly under the use of the proto-carbonate of iron.

I cannot doubt that the musical murmur in both these cases had its seat in the artery, and that they were examples of local arterial pulsations, with extraordinary murmur, unattended by organic disease.

Dr. Hope has given what he terms a new presumptive sign of aneurism in cases where the tumour lies immediately behind the heart, and pushes this organ forward. There is then, according to him, the double-jogging impulse similar to that produced when the heart is thrown in front of the spine, or bound down by pericardial adhesions. He also states, that when this double impulse is accompanied by a murmur, which we can clearly determine not to be valvular, the diagnosis of aneurismal tumour lying behind the heart may be made. In a case recorded by him the impulse of the heart was exceedingly vigorous, double, and consisting of a diastolic as well as a systolic impulse, each of a "jogging" character. All who saw the patient agreed that there must have been considerable hypertrophy to account for so strong an impulse, yet the heart was found but slightly enlarged, the left side being a little thickened, while the right cavities were dilated without any increase of the muscular substance.

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[1] See a case of Constriction of the Aorta, by Dr. Nixon, Dublin Medical Journal, vol. v., in which a small abdominal tumour existed giving pulsation and musical murmur. This disappeared before death. Its cause was never discovered.

Dr. Hope, however, admits, that when the pressure is very great, the sounds of the heart may be diminished. This was observed by Dr. Todd in a case where the heart was pushed forwards and outwards, and compressed against the ribs by an enormous aneurism of the thoracic aorta. In this case the sounds of the heart were so modified as to lead to the diagnosis of concentric hypertrophy.[1]

We are not, however, justified in admitting the double-jogging impulses of Dr. Hope, with or without murmur, as indicative of aneurism behind the heart. The diagnosis from these signs would be very uncertain, even though there were no evidences of pericardial adhesion or displacement of the heart to the front of the spine. What Dr. Hope means by the jogging impulses is not very clear, but we know that a distinct double impulse of the heart, systolic and diastolic, may occur without the existence of aneurism, or, indeed, of any disease of the circulating system whatever. We cannot concur with Dr. Hope when he speaks of a murmur in the præcordial region *distinctly not referrible to a valve*, but we may admit his statement, that "if there be no murmur whatever in the præcordial region, yet one audible on the back, the evidence of aneurism afforded by this sign, in connexion with the double jog of the heart, and the posterior dulness on percussion, is almost positive."

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[1] "I can easily believe," says Dr. Hope, "that if the compression be very great, the sounds may be diminished; for in my first experiments on the denuded heart of the ass, I have found that heavy pressure with the stethoscope on the ventricles invariably diminished the sounds, of course, by curbing the contractions of the organ and the extension of the valves. It remains, therefore, to be ascertained, by further cases, whether diminution of the sounds will prove to be a constant sign of an aneurism behind the heart occasioning great pressure."—*Op. Cit.*

DOUBLE SOUND AND IMPULSE IN ANEURISM.

We have already dwelt on the fact, overlooked by Laennec, that, in thoracic aneurisms, the existence of a first and second sound, very similar to those of the heart, was a common occurrence. The distinctions, then, which Laennec has given between the sounds of aneurism and of the heart, founded on the singleness of the aneurismal sounds, cannot be admitted. This doubling, not only of the sounds, but of the impulse of aneurism, was noticed by me in 1833, and in the following year I published a case of aneurism of the innominata, which, so far as I know, is the first instance in which these phenomena were recorded; since then the question has been handled by M. Guerin and by Dr. Bellingham; the last of whom has contributed an extended series of researches on the subject.[1]

Some of these latter observations are applied by the author in considering the theory of the sounds of the heart. With respect to aneurism, however, the following conclusions have been arrived at by Dr. Bellingham:—

1. That a double, not a single, sound characterizes aneurism of the arch of the aorta, which closely resembles the double sound of the heart, and may be termed its normal sound.
2. That the normal double sound of aneurism of the arch of the aorta, has its cause in the friction between the blood and the lining membrane of the orifice and parietes of the sac, because there is no other agency to which it can be referred.
3. That the normal second sound of aneurism of the arch of the aorta, is caused by the regurgitation of the blood into the sac from the aorta and large vessels which arise from it.
4. That the first, or the second, or both, aneurismal sounds

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[1] Dr. Bellingham's memoirs are to be found in the nineteenth volume of the Dublin Medical Press, 1848.

may be replaced by a murmur, which may have either a blowing, sawing, or filing character; and that such murmurs may be regarded as the *abnormal* sounds of aneurism of the arch of the aorta.

5. That the first aneurismal sound is much more frequently superseded by a murmur than the second,—because the force with which the blood is transmitted to the sac is much greater than that with which it regurgitates into the sac at the period of the ventricular diastole.

6. That the abnormal sounds of aneurism of the arch of the aorta, equally as its normal sounds, are caused by friction between the blood and the orifice or parietes of the sac, and that they are nothing more than exaggerated normal sounds,—exaggerated because the degree of friction is then increased.

7. That in aneurism of the arch of the aorta, pointing externally, the sound is not always double, but a double impulse is frequently also perceptible to the hand.

8. That the second impulse of aneurism of the arch of the aorta has its cause in the same agency which gives rise to the second sound; consequently neither a double sound nor a double impulse are perceived in aneurism of the abdominal aorta, or of any of its branches.

9. That the phenomenon known under the name of *frémissement cataire*, or purring tremor, whether it occurs in an aneurism or a large artery, is nothing more than the pulse of aortic regurgitation on a large scale; consequently that it is a sign of regurgitation into the ventricles of the heart, into an aneurismal sac, or into a large or a dilated artery.

10. That the remarkable resemblance between the normal and abnormal sounds of aneurism of the arch of the aorta and the normal and abnormal sounds of the heart, renders it probable that the mechanism of their production is the same.

It will be seen that Dr. Bellingham explains the second impulse and sounds of thoracic aneurisms, by reference to regurgitation into the aneurismal sac from the large arteries given off by the arch; an explanation which is consistent with the fact that the second impulse and second sound, distinct though they may be, are feebler and shorter than the first. In his view, then, the cause of these phenomena is not an active one; it is simply a reflux of a certain portion of blood during the ventricular diastole, and does not imply any independent vital or elastic action of the sac, or any vital force given by the artery. The weight of the regurgitating column is the sole cause. And he explains the absence of double sounds and impulse in abdominal aneurisms, by the want of a mechanism similar to that in the case of aneurisms of the arch of the aorta.

A different explanation is given by Dr. Lyons of these phenomena, which, according to him, are to be attributed to the action of two independent and active forces,—one the ventricular systole causing the first impulse and sound, and the other the systole of the artery,—which sends forward a second wave of blood less strong than the first. He holds that for the production of the second sound or impulse a certain form of the sac is necessary, and that the chances of a double sound are directly as the sphericity of the tumour or sac. Hence the sign in question is common in false aneurisms. Under these circumstances the second impulse given by the arterial systole is rendered more manifest by the operation of the hydrostatic law. But in aneurisms of an elongated fusiform shape, and especially when the middle coat is unbroken, as in true aneurisms, the divergence of the sides being little, the walls of the aneurism re-act simultaneously with those of the artery, and we thus lose the second impulse. The second sound may or may not be present; its production is to be considered as dependent on the state of the internal surface of the vessel.

In order, however, to clear up this subject, an extended series of observations on the signs of true aneurisms as compared with the false, and also on the law which governs the occurrence of double pulsations in arteries unaffected by any form of aneurism, is still required.[1]

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[1] On the Motions and Sounds of Aneurism, by Robert D. Lyons, M. B., Dublin Quarterly Journal of Medical Science, vol. ix. 1850. Both Dr. Lyons and Dr. Bellingham agree as to the occurrence of a single sound in abdominal aneurisms. To this point we shall recur when speaking of that disease. The following observations of Dr. Lyons are important as bearing on the question before us.

"If, therefore, true aneurism be less likely to assume the spherical figure than false, the absence of the second impulse may, perhaps, be available in approximating to a diagnosis between these two forms of the disease, especially if com-

Some observations on the effect of position in modifying the sound of aneurisms have been made by Dr. Lyons. In a case of thoracic aneurism, with double sound, and double impulse, it was found that when the patient was placed in the recumbent position, the head being on a level with the trunk, the double impulses and sounds were as fully developed as in the upright posture. In another case,—one of traumatic varicose aneurism of the femoral artery,—the impulse was distinctly single, and unaffected by elevating the foot, so as to allow the column of blood naturally below the sac to be then above it.

bined with the observations of Stokes and Gendrin as to the less degree of suffering in the true. The explanation I have given of the impulses and sounds of thoracic aneurism agrees very nearly with that of Gendrin, who, however, states that they are to be found in the abdominal form also. I know, however, of no recorded case of double impulse of abdominal aneurism, though I have alluded before to some cases in which a double sound was heard. An explanation of the absence of the second impulse, and, except in rare cases, of the second sound, in this form of the disease, may be deduced, I think, from the following considerations.

The column of blood in the arch and superior part of the thoracic aorta must be considered as subject to two forces acting at an appreciable interval, viz., the ventricular and arterial systoles, whose influence must be supposed to be combined and simultaneous on particles of blood at a distance from the heart; for in no other way can we explain the single pulsation of the carotid, femoral, and radial arteries. The sequence of actions may be considered to take place as follows. The blood contained in the arch of the aorta, receiving an impetus from the left ventricle, is set in motion towards the extremities, but, before it can be propelled to any great distance, it receives the additional impulse of the systole of the first portion of the aorta, and now continues its course impelled by the combined influence of two forces, that of the ventricular systole being, as it were, overtaken by that of the arterial. It is highly probable that these forces become thus simultaneous in their action in some part of the descending thoracic aorta, and that the ordinary impulse of the abdominal aorta and its branches, as well as that of the carotid, radial, and femoral, is single. Indeed that of the ascending portion of the arch is most probably single also, and it is only when a laterally divulging current is produced into an aneurismal sac, that the arterial systole manifests itself by giving a second impulse. The systole of the arteries here mentioned is caused by the elasticity of their walls. If their contractile powers should be brought into action by deranged nervous agency, the phenomena of local arterial pulsation, dicrotous pulse, &c., can be explained. The single impulse of abdominal aneurism, therefore, may, I think, be justly referred to the combined and simultaneous action of the ventricular and arterial systole; while the occurrence of two sounds may be accounted for, the first and normal one by the entrance of the blood, the second, so rarely heard, by the egress, produced by an elastic state of the sac."—*Op. cit.*

But we should not adopt either of these explanations of the double impulse and sounds in thoracic aneurisms in an exclusive manner. Nor can we go to the full length with Dr. Lyons, in declaring that the theory of regurgitation, as given by Dr. Bellingham, is untenable. I believe that we may have regurgitation into an aneurism, though it is still to be proved that this can cause a second impulse or shock.

It is now some years since I exhibited at the Pathological Society a specimen of true aneurism of the ascending aorta. The patient presented the visibly throbbing arteries, so characteristic of regurgitation through the aortic opening, and yet the valves were not patent. On that occasion I suggested that in certain cases, regurgitation through the permanently patent aneurismal orifice might produce the appearance in question.

It appears to me, also, that both causes may act in producing the second impulse and sound, for the moment of regurgitation and of arterial systole cannot be very different. Both regurgitation and arterial systole must occur in the intervals of ventricular systole. And it is difficult to understand how the arterial systole can give a distinct second impulse when the aneurism exists at the very origin of the aorta.

A double action in arteries, when in a state of excitement, bearing the closest analogy to the double action of aneurisms, is often met with. In these cases the conditions of regurgitation may be absent; and the double impulse and sound can only be explained on the principle of the diastolic wave being followed by arterial systole. This fact deserves consideration, more especially when associated with the observation of Dr. Lyons, which I can fully confirm, that in many cases of double impulse and double sound in aneurism of the aorta, the appearances of regurgitation are wanting in the vessels of the neck and upper extremities. In truth, the combination of a doubly impulsive and doubly sounding aneurism, with the signs of arterial regurgitation, is the exceptional case.

It is not yet established that regurgitation is accompanied by any impulse when it is the result of gravity, as it should be in an aneurism. On Dr. Bellingham's view of the second sound, there is an ascending and a descending column; but though we have a cause of shock for the ascending column, namely, the ventricular systole and arterial contraction, we want this for the descending column, unless the elasticity of the arteries of the neck acts on the fluid. Murmur, indeed, might be produced, and we might expect that in the doubly sounding aneurisms the to-and-fro murmur, so common in regurgitant disease of the heart, should be often met with, but unquestionably this is not the case.

Again, in ordinary cases of regurgitation, where the aortic valves are permanently open, the descending column, though producing a murmur, gives no shock like the second impulse of an aneurism. The phenomena in this case are, a strong and extended systolic impulse, and a to-and-fro murmur behind the sternum. Double impulse of the heart, in its ordinary form, is rare. When it occurs, the second impulse occupies a situation different from the first. I know of no instance of permanently open aortic orifice where two impulses occupying *the same situation* have been observed in the left ventricular region. If, then, the regurgitation into the ventricle does not produce a distinct impulse—if a column of blood, coming from the aorta into a flaccid ventricle, only causes murmur, it seems improbable that the regurgitation from the arteries of the neck could cause a shock or impulse in an aortic aneurism.[1]

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[1] Referring to M. Guerin's views as to the cause of the second sound in aneurism, Dr. Bellingham observes as follows:—"With respect to the second sound heard in aneurism of the arch of the aorta, this had been long since noticed by Dr. Stokes as not an unfrequent phenomenon in aneurism of this part of the artery, but the mechanism of its production was not known. M. Guerin, in the 'Revue Medicale' for the year 1844, was the first who offered anything like a satisfactory explanation of it; he was likewise the first to call attention to the second impulse in aneurism of this vessel. I had, however, been familiar with the latter before I had seen M. Guerin's communication."—*Medical Press*, vol. xix. page 378. In my paper on the Diagnosis of Aneurism, published ten years before the appearance of M. Guerin's researches (see the Dublin Medical Journal, 1834,) I have given a case of a true aneurism of the ascending aorta in which the double impulse was observed. It disappeared under a reducing treatment, though the double sounds continued, and reappeared after an attack of severe pain in the chest. The fact of double impulse in aortic aneurism was published by me in 1834, and again by Dr. Greene, in 1839. Dr. Bellingham's researches appeared in 1848. In these memoirs I find that the author, in speaking of the similarity of the sounds of the doubly beating aneurism with those of the heart, comes to the following conclusions—among others on the same subject:—

"10th. That the remarkable resemblance between the normal and abnormal

Modification of signs in varicose Aneurisms.—The various works on aneurism abound with examples of perforations, which may be divided into those where the blood returns into the circulating stream, and those in which it is forced into a new cavity. The term of communicating aneurisms may be applied to the first form, of which many examples are recorded. Thus we may find perforations into and communications with the right or left ventricle, with the pulmonary artery, the vena cava, or even the thoracic duct.

We owe to Mr. Thurnam the most important memoir on this subject;[1] and when the number of cases which he has collected is considered, we must agree with him in the opinion, that spontaneous varicose aneurism is not to be considered as a mere pathological curiosity, but, on the contrary, a lesion with which the practitioner should be prepared to meet. In a practical point of view, the characteristic of the disease is the existence of a communication between the currents of arterial and venous blood, and the opening may be into the right ventricle, the right auricle, the pulmonary artery or vena cava. And the symptoms which result from the forced admixture of arterial with venous

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sounds of aneurism of the arch of the aorta, and the normal and abnormal sounds of the heart, renders it probable that the mechanism of their production is the same.

"14th. That as sounds, almost precisely similar to those of the heart, are developed in an aneurismal sac which has neither muscular walls nor a valvular apparatus at its orifice, the latter do not appear to be as essential to the production of the normal sounds of the heart as most writers suppose."—*Medical Press*, 1848.

In my paper on aneurism, I observed, "that the occurrence of double pulsations, similar to those of the heart, seems to prove that the re-action of a single cavity may, under certain circumstances, produce a double sound, and hence it appears probable that the division of the heart into auricle and ventricle is not necessary for its double sound. The explanation of the sounds of the heart, founded on this division, must be reconsidered; and it is not unlikely that mere systole and diastole of a single cavity, or of two cavities acting synchronously, as in the case of the ventricles, with the entrance and exit of a fluid, are all that are necessary for the production of the second sound."—*Dublin Journal of Medical Science*, vol. v. 1833.

These observations may possibly have escaped Dr. Bellingham's notice. But I do not believe that, in explaining the cardiac sounds, we can neglect the action of the valves.

[1] Medico-Chirurgical Transactions, Second Series, vol. xxiii.

blood, added to those of arterial disease, are what might be expected from this complicated lesion. Mr. Thurnam has especially noticed the rapidly extending anasarca of such portions of the body as are distal to the varicose orifice. The distention of the hepatic and sub-cutaneous veins follows the same rule. The pulse is jerking, and in some cases very feeble; and the patient complains of debility and chilliness. Dyspnœa, orthopnœa, and, finally, apnœa, occur.

The physical signs noticed by Mr. Thurnam are principally "the superficial, harsh, and peculiarly intense sawing or blowing sounds, accompanied by an equally marked purring tremor heard over the varicose orifice, and in the current of the circulation beyond it. This sound is continuous, but is loudest during the systole, less during the diastole, and still less during the interval."

The most important sign is the thrill or purring sensation of aneurismal varix, which, when predominating along the right border of the sternum, indicates an opening into the inferior cava or right auricle, but when on the left, into the pulmonary artery or right ventricle. The occurrence of the preceding symptoms and signs following on an unusual effort, would make the evidence of varicose aneurism almost indisputable.[1]

Dr. Hope has recorded two remarkable cases of communication between an aneurism of the aorta and the right ventricle in one instance, and the pulmonary artery in the other. In both instances the physical signs were accurately observed, but as the patients did not come under observation until after the false passage had occurred, there is still some doubt as to the absolute value of the observed signs as diagnostic of perforation. In the first case, which was observed by Dr. Hope, the symptoms set in after an exertion in lifting a heavy weight, when the patient felt a "*creak in the heart,*" and became faint and very pale. The second case was that of a porter, who had been about ten months before attacked with pneumonia, followed by the usual symptoms of heart disease. In this case a communication was found between the aorta (which had been dilated) and the pulmonary artery, but the exact period at which this perforation took place was not marked by any new or distinct symptom. In both these cases the pulse was pre-eminently jerking, with a marked superficial murmur accompanying both sounds, certainly continuous in one case, and probably so in the other. This was associated with a purring tremor, existing from the level of the fourth rib up to the interspace between the second and third in the first case, and between the second and third only in the latter. There was in both great, rapid, and universal dropsy, and the complexion was livid.

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[1] See Dr. Hope's work, fourth edition, page 439.

A case of varicose aneurism by Professor Smith is too important to be omitted here. The communication existed between the aorta and pulmonary artery.

A young and muscular man, who had always been healthy, became subject, about three months before his death, to attacks of vertigo, with temporary loss of vision. He felt weak, and was unsteady in walking, particularly after any excess, when he could hardly keep himself from falling in the street. He was always pale and chilly, and the fits of weakness were so great that he had frequently to stop and lean on the nearest object for support, and was twice brought home fainting. On admission into hospital he was found anasarcous; face pale and bloated; dyspnœa; orthopnœa; the lips blue, and the pulse variable and intermitting. The patient suffered from frightful dreams and great anxiety. The heart's action was strong and tumultuous, and the sound on percussion dull from the second to the eighth rib. A loud bellows murmur accompanied the first sound, and an intense purring tremor could be felt over the whole cardiac region. When the bellows murmur became less loud, this tremor increased. Nausea, vomiting, headach, and fits of weakness, terminating in complete syncope, supervened, and the patient had attacks like epileptic paroxysms, but he did not froth from the mouth. The purring tremor increased. There was pain in the region of the heart, frequent sense of suffocation, and inability to lie on the right side. He was most anxious to rest, but afraid to sleep lest he should be suffocated. He complained of a fluttering sensation, as if, to use his own words, there was a living bird within his chest. The purring tremor increased to such a degree that it could be felt through the bed-clothes, and the sound heard at a great distance from the patient. All these symptoms increased in violence. The action of the heart continued strong and tumultuous, but the pulse was weak, and the tremor still more violent. He died on the day following one of the pseudo-epileptic fits.

The auriculo-ventricular openings were healthy, the left ventricle dilated, and the right perhaps a little thickened. Both cavities were filled with dark, fluid blood. The pulmonary artery communicated with the aorta by a small opening at the origin of the latter vessel. The edges of this opening were thickened and rounded off. There was dilatation of the aorta at the point of opening, with distinct signs of arteritis.[1]

It will be seen that in these cases there is a combination of physical signs which does not belong to any ordinary case of disease of the valves, and that where these signs and symptoms suddenly occur after a violent effort, the diagnosis of a communicating aneurism may be made with considerable probability. This diagnosis would be greatly strengthened if an opportunity had occurred of examining the patient previous to the supervention of the new phenomena.

It must, however, be admitted, that the diagnosis not only of these communicating aneurisms, but of the dissecting variety, is still imperfect; yet in most of such cases, evidence of important organic disease will be sufficiently manifest, and with this, in the present state of our knowledge, the practical physician must rest satisfied.

In these cases the patient dies with the symptoms of gradually obstructed circulation, and not, as in most other instances, suddenly, in consequence of the loss of a great quantity of blood. When the aneurism opens by a sufficiently large orifice into the trachea, œsophagus, or one of the three thoracic serous cavities, sudden death is the ordinary result. Indeed, some years ago, we believed that such a result was inevitable, but later experience has established the fact that, in certain cases, the hemorrhage may take place, not only into free serous cavities, but through the integuments,—by successive gushes, between the periods of which complete re-action may occur.

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[1] See Dublin Journal of Medical Science, vol. xviii. p. 164.

COMPRESSION OF THE TRACHEA OR BRONCHIAL TUBES.

When an aneurismal tumour comes into contact with the trachea, or one of its divisions, two remarkable phenomena may be produced. Of these the first, or the stridor, has been long recognised; while the second, namely, the production of an inequality of respiration,—as shown by the difference in strength of the vesicular murmur in either lung,—has not received the attention which it deserves. When the clinical history of aneurism is considered more at large, we shall recur to these signs, and at present content ourselves with a brief notice as to their nature and value.

The phenomena in question may be thus enumerated:—

1. Tracheal stridor.
2. Tracheo-laryngeal stridor.
3. Bronchial stridor.
4. Diminished vesicular murmur in one lung.

The different examples of stridor may occur conjointly, or in an isolated manner. We give the name of tracheal stridor to the character of respiration induced by a simple narrowing of the trachea, which may arise from pressure, independent of any obstruction at the top of the windpipe. This is more likely to occur where the pressure is lateral than where it is directly from before backwards, so that a small aneurism, making lateral pressure, may cause more distress than a large one lying in front of the trachea. In these cases the stridulous sound is commonly produced at a point below the region of the larynx. To this I have given the name of *the stridor from below.* But where there is a complication with irritation or spasm of the larynx, this character is not so easily determined; and to this form we may give the name of the tracheo-laryngeal stridor.

The third form, or that of bronchial stridor, is met with in cases where an aneurism lies on one of the main bronchial divisions. And yet, although the trachea is not compressed, we may often detect a feeble stridor from below. This is a curious and not often recognised sign, and it passes into true tracheal stridor according as the tumour increases in size.

Lastly, the diminution of respiration in one lung must be taken as a sign of great importance. It may be met with in a well-marked form, where the aneurism is so small as to give neither stridor, distinguishable pulsations, or dulness on percussion. I think that the right bronchus is more often the seat of compression by aneurism than the left.

Under these circumstances we have the existence of feebleness of respiration in one lung, unexplained by any previous or existing pulmonary disease. The sign is, as it were, isolated, and neither percussion nor the stethoscope detects disease in the lung.

When the natural difference of the vesicular murmur in the lungs is borne in mind, it will be obvious that its feebleness at the left is more valuable than at the right side. Yet even on the right side we may find a degree of feebleness of respiration, even more than natural, indicative of the pressure of a tumour on the bronchial tube.

Careful comparison of the respiratory murmur in both lungs must be made in every case of suspected thoracic aneurism. The feebleness of respiration is, in most cases, general over the lung, and proportionate to the narrowing of the tube.

This combination of general feebleness of respiration in one lung, without any evidence from percussion of disease in the pulmonary structure or pleura, and without signs of bronchitis as in Laennec's emphysema, occurs, so far as I know, in but one other case, and that is, where a foreign body partially obstructs one of the main bronchi. Hence it may be laid down that, in a case in which there is no reason to suspect such an accident, the combination in question would be diagnostic of the pressure of a tumour on the bronchial tube. Of course, where the tumour is of sufficient size, it may cause a corresponding dulness in the interscapular region, as occurred in a case recorded by Dr. Hope, in which, notwithstanding this dulness, the absence of signs of pulmonary or pleural disease led to the suspicion that an aneurism existed.

CLINICAL VIEW OF ANEURISM.

Under this head may be examined the varying symptoms of the disease in its different forms, its mode of termination, its combinations, and the principles which guide us in its treatment. So far as the stethoscope is concerned, we have spoken of the two great sources of diagnosis, namely, the acoustic phenomena which belong to the disease itself; and again, those which result from the compression of the tumour on some part of the respiratory apparatus. But in dealing with the subject at large, we must again and again return to the study of both these classes of physical signs.

Aneurism of the thoracic aorta does not present any special symptoms, and occasionally may be one of the most latent of diseases; its symptoms, such as they are, are anything but constant, and this we should expect when the varied sources of these phenomena are considered. Indeed, with the exception of pain, there appears to be no symptom proper or essential to the disease; and this pain is of uncertain occurrence, and varies in its seat, nature, and amount. The remaining symptoms are referrible to disturbance of the pulmonary and circulating systems, or to the simple effect of pressure on parts such as the œsophagus, air-passages, nerves, or blood-vessels.

We may, then, divide the evidences of the existence of aneurism into three heads:—

1. Pain.
2. Symptoms of disease of the circulating and respiratory systems, which are often unattended by positive evidence of disease in the lung or heart.
3. Signs of intra-thoracic tumour.

It need hardly be stated, that the last set of phenomena are common both to cancer and aneurism. We shall presently examine whether any or what difference exists between the signs of these forms of tumour.

If, with reference to pain, we compare thoracic with abdominal aneurism, it will be found that in the latter disease it is more frequent, and often more intense. Indeed, pain of a peculiar character is one of the great symptoms of abdominal aneurism, while its occurrence when the disease is situated in the thorax is inconstant. We may often diagnosticate an abdominal aneurism from the character of the pain; but this is seldom the case when the tumour is situated above the diaphragm.

On the other hand, if suffering, independent of pain, be considered, patients with thoracic aneurisms are generally more exposed to distress of some kind. There is often more constitutional suffering, especially in the case of false aneurism; cough, dyspnœa, and palpitation, are frequently present, or easily excited, and where pressure exists on any important organ, the distress is more or less constant.

It may be, that a cause of the lesser frequency of pain in thoracic, as compared with abdominal aneurisms, is the greater frequency of true aneurism within the thorax. And there is nothing more singular than the absence not only of pain, but of the signs of compression of surrounding parts which is occasionally met in true aneurisms of the thoracic aorta. Of this the following is a good illustration:—

CASE LXX.—*Symptoms and signs of chronic Phthisis; Absence of physical signs of Aneurism; Tussis Clangosa; Vast multilocular Aneurism of the arch of the aorta.*

A gentleman, past middle age, after repeated exposures to cold, was attacked with cough, having a loud, ringing character. After some time he consulted Sir Philip Crampton, who came to the conclusion, that he was threatened with tubercle, and advised a change of climate. Not satisfied with this opinion, he placed himself under another practitioner, who declared that the symptoms proceeded from hepatic disease, and advised a course of mercury, after which there was a great diminution in the severity and frequency of the cough. He then resumed his usual habits; but the cough returned; and after a period of six months he again used mercury, but without the same relief as before. Constitutional symptoms now began to show themselves; but the cough was still dry. It was a single cough, having the metallic, ringing character. The symptoms had now existed for more than a year, when he again placed himself under Sir Philip Crampton's care. The signs of tubercle of the right lung were now no longer doubtful, the clavicle was dull, while a deep-seated muco-crepitating râle occupied the upper lobe of the lung. Hectic fever set in, and the patient began to expectorate. At this time I saw him along with Sir Philip Crampton. The cough had become loose and paroxysmal; but at the termination of the fit, a single sonorous inspiration, somewhat similar to the whoop in pertussis, but having its seat evidently under the sternum, could be perceived. Neither dysphagia nor permanent stridor existed. The radial pulses were equal, and, with the exception of the peculiar sound above described, there was nothing to lead to the suspicion of aneurism. A few days before his death we discussed the probability of the existence of enlargement of the bronchial glands.

On dissection both lungs were found to contain a great quantity of tubercle. The deposit at the right lung partook more of the character of tubercular infiltration; its upper lobe presented an anfractuous cavity. The left lung presented numerous isolated tubercles. On pressing the lower lobe, it was found to contain numerous spiculæ, which proved to be bronchial tubes transformed into earthy structure of extreme hardness.

A vast aneurismal dilatation, engaging the whole ascending portion and arch of the aorta, was found; it was fully four inches in breadth, and more than six in length, and presented two or three pouches containing a quantity of dense and laminated fibre. One of these cells, fully as large as a hen's egg, lay exactly in front of the trachea; it was completely filled with fibrine. The innominata, the left carotid, and subclavian arteries, were free from disease.[1]

We shall hereafter examine how far it is possible to distinguish during life between a false and a true aneurism.

We have seen, that although pain is not unfrequently present, it is by no means so prominent a feature as in aneurisms of the abdomen. It is seldom severe; it is of an uncertain and fugacious character, and is often removable, at least for a time, by treatment the reverse of active. It has more the characters of neuralgic than of inflammatory pain, and may be felt in various situations, of which the most common are, the shoulder, the side, the neck, or sometimes deep in the cavity of the chest itself (shooting from the upper third of the sternum towards the spine.) It often affects the arm, or extends upwards along the neck to the side of the head. In a case lately under observation, the patient chiefly complained of the pain in the right side of the head. These pains are sometimes augmented in a deep inspiration or other motions, and Dr. Greene has noticed that they are occasionally relieved by pressure.[1]

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[1] See the Transactions of the Pathological Society. Dublin Journal of Medical Science, vol. xv. page 154.

The occurrence of a boring pain, generally constant, and with exacerbations, has been attributed to erosion of the vertebræ. But this opinion must be received with caution, for in abdominal aneurism, at least, we may have erosion of the vertebræ without pain, and pain without any injury of the spine: indeed, the diagnosis of aneurismal erosion of the spine is still uncertain, although the occurrence of the lesion may be considered probable in cases presenting the two distinct forms of pain, as indicated by Dr. Law,—one, the lancinating, paroxysmal, and remitting pain; the other, the constant, dull, boring sensation, confined to a certain locality.

When the bodies of the vertebræ are destroyed to a great extent, the patient may suffer from pressure exercised on the aneurismal tumour itself by the superincumbent weight; and thus we obtain an indication of absorption of the vertebræ. We shall presently study a case in which the usual symptoms of internal pressure, such as dysphagia, dyspnœa, and cough, were absent while the patient was supported on crutches. But the attempt to stand without the crutches brought on those symptoms. In this case, too, there were other remarkable circumstances. Great relief was given by the use of issues; and there was unequivocal evidence that, as the disease advanced, the tumour retreated from the anterior portions of the chest.

In some cases, however, the pain, from its severity, becomes a prominent symptom, and is felt in the shoulders, shooting down to the sides and catching the breath; or it may occur in the interscapular region, extending to the neck and head, with a sensation compared by the patient to that which would be caused by pouring hot water down the back. All these forms of pain may be attended with numbness of the distal nerves, with a sensation of weight and heat in the chest, or with tenderness of the surface on pressure.

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[1] Researches on the Symptoms and Diagnosis of Aneurismal and other Tumours in the Cavity of the Thorax, by George Greene, M. D. Dublin Journal of Medical Science, vol. x.

It is yet to be determined how far the occurrence or character of pain will enable us to distinguish between a true and false aneurism of the aorta. Pain is probably much less frequent in the former than in the latter variety. I have recorded a case of true aneurism, engaging the whole course of the aorta to the origin of the innominata, in which severe pain existed for about four weeks previous to the external appearance of the tumour. On one occasion the pain was attended by numbness of the left arm; death took place by rupture into the pericardium.[1]

We are not, however, justified in connecting these aneurismal pains with the absorption or perforation of surrounding parts,—processes which often occur, as it were, silently, and of which the first evidence is a gush of blood in some new direction. Finally, we observe pain connected with irritation of the pleura, as is shown, not only by dissection, but by the existence, as I once observed, of a pleuritic friction sound confined to the region of the aneurism.

SYMPTOMS OF COMPRESSION.

Under this head are to be enumerated the most important symptoms of aneurism, which, however, it has in common with other forms of intrathoracic tumour. Of these tumours, it may be laid down that aneurism and cancer are the most frequent examples; so that an intrathoracic tumour having been discovered, the diagnosis is in most cases to be made between these forms of disease. To this subject we shall return.

The parts whose compression gives rise to important symptoms are—

1. The trachea or one of its primary divisions.
2. The œsophagus.
3. Portions of the arterial and venous systems.
4. The eighth nerve.
5. The lung.

Let us consider these examples of pressure in a clinical point of view.

Compression of the air-passages.—It has been shown, that the pressure of an aneurism may cause a narrowing of the trachea, which produces the stridor from below. It is of great importance to familiarize ourselves with this sound, as it happens too often that this stridor is taken to indicate chronic diseases of the larynx, an error involving the worst consequences in treatment and prognosis. In most cases a little attention will show us that the sound issues, as it were, from the fourchette of the sternum: and this is manifest, even without the aid of the stethoscope; so that the experienced observer, on seeing the patient for the first time, even when standing at a distance, can declare that the seat of stridor is not in the larynx. The amount of stridor varies greatly in different patients; and in the same individual at different times; generally it is greatest after exertion; and sometimes requires a deep and forcible inspiration to produce it.

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[1] See my Memoir on Aneurism, Dublin Journal of Medical Science, vol. v.

It is common to all forms of intrathoracic tumour, and theoretically should be induced by simple contraction of the trachea; but of this I have not seen any instance.

The liability to its production seems to depend much more on the direction of pressure than on the nature or even the size of the aneurism. It is met with in true as well as false aneurisms, but is probably more common in the latter. If pressure is exercised on the side of the trachea, a very small aneurism may cause the symptom. When we reflect on the mechanical structure of the trachea, we can understand that the ends of the cartilaginous bodies next to the membranous portion will yield much sooner than their centres, which form the apex of the arch. And thus it happens, that the entire form of the tube may be altered by a folding of the trachea on itself, sometimes so great as nearly to make it represent a double tube. This occurred in a case of aneurism of the innominata, in which the pressure was so extreme that the free ends of the cartilages on one side overlapped those of the other so as almost to prevent the entrance of air. So great was the displacement, that the centre of the thyroid cartilage corresponded to a line drawn from the ramus of the left jaw to the humeral extremity of the clavicle.[1]

In certain cases the walls of the trachea may be so compressed as to give way by ulcerative absorption or by sloughing; this occurred in a case observed by Professor Smith, in which the aneurism had perforated one side of the tube, and so compressed the other, that it was adherent to the coagulum, and showed a circular patch of sloughy structure.

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[1] Researches on the Diagnosis and Pathology of Aneurisms, Dublin Journal of Medical Science, vol. v. 1836.

A most singular circumstance in this case was the absence of stridulous breathing. There was but little dyspnœa. The voice became weak; and the patient was aphonic for about six weeks before death, during which time he was paralyzed on the right side, and had frequent epistaxis. The respiratory murmur was less distinct in the right than in the left lung.

Hence it appears that, in aneurism of the aorta, we may have aphonia without stridor, notwithstanding the existence of great compression of the trachea. The occurrence of stridor without aphonia is much more common.

But the aneurismal stridor is not in every case so well-marked as to be easily distinguished, at least by its own character, from that of chronic laryngeal disease. We have already spoken of this form under the head of tracheo-laryngeal stridor. It is more common when there exists a complication with bronchial disease, and may proceed either from a co-existing spasm, or from actual change of the larynx.

Of these causes, the first is probably the most frequent; and this opinion is strengthened by the fact, that in certain cases of aneurismal pressure, the stridulous breathing has been relieved for a time by tracheotomy. We have seen that in Dr. Smith's case there was no stridor, notwithstanding the great narrowing of the trachea; and all these circumstances point to the conclusion, that the stridor in aneurism, like the alterations of voice, is more or less under the influence of deranged innervation.

I have observed the bronchial stridor in several cases, but it is not often sufficiently distinct to enable us to localize it. It may be caused by aneurism of the innominata, or by an acute and very copious empyema. This stridor does not necessarily exist where there is inequality of vesicular murmur.

Closely connected with these forms of stridor is the sign of inequality of respiration in the lungs, to which I drew attention many years since.

I have never met with such an occlusion of the bronchial tube as to cause total suspension of respiratory murmur throughout the lung; but we may well conceive its possibility. It frequently happens, however, that the pressure is sufficient to induce great feebleness of murmur on the affected side, while the opposite lung exhibits permanent and intensely puerile respiration. We have alluded to the resemblance of these signs to those of foreign bodies in the air-passages. The first case, or that of perfect occlusion of the bronchial tube by pressure, would resemble that of the complete stoppage of the tube by a smooth, foreign body, such as a kidney-bean; while the more ordinary instance of a generally diminished murmur would resemble that in which an irregular body, such as a tooth, while obstructing the passage, yet permits the entrance of air.

We have met with no case in which the diminution of the murmur was not general over the lung; yet should the pressure be only exercised on the second division of the tube, partial feebleness of respiration may be found.

The first case in which these phenomena were observed occurred to me in 1833.[1] There was no proper physical sign of aneurism; the respiration in the right lung was generally feeble; and dysphagia with irritability of the stomach were the principal symptoms.

This feebleness of respiration generally remains constant; it is possible that by the advance of the tumour all vesicular murmur might be suspended. On the other hand, a change in the direction of pressure might so relieve the bronchus as that full respiration would return over the lung.

We have yet to note some additional phenomena proceeding from pressure on the bronchus. These are,—

1. Obstruction of the tube, causing absence of murmur in the lung during the first half of the period of inspiration.

This was observed in a case where an aneurism of the descending aorta compressed the left bronchus. On applying the stethoscope to the left axilla, no sound was heard during the first half of inspiration; but during the latter half the air suddenly rushed in as if a valve had been opened. This observation I made repeatedly, and with care.

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[1] This case has been published by Dr. Porter, Professor of Surgery to the Royal College of Surgeons in Ireland. See his Cases of Internal Aneurism, Dublin Journal of Medical Science, vol. iv.

2. Comparative fixity of one side of the chest during inspiration, with increased expansion of the opposite side.

3. Absence of vocal vibration in the upper portion, and great feebleness of vibration in the lower part of the side, while it was strongly marked in the opposite lung.

All these phenomena, which are what we might expect in a case of great compression of the tube, were observed in the one patient.[1] The left bronchial tube was found considerably narrowed and indented, and a perforation, communicating with the aneurism, existed. The patient died from hemorrhage.

Dr. Greene has recorded a case in which unequal dilatation of the side during inspiration was observed.[2]

But the most important example of the effects of this condition is that given by Dr. Mayne.

Case LXXI.—*Aneurism of the transverse portion of the Arch of the Aorta, extending to the left side; Contraction of the side, similar to that following on the absorption of an empyema; Displacement of the Heart towards the Axilla.*

"Dr. Mayne presented a specimen of aneurism arising from the front of the transverse portion of the arch of the aorta, and extending over to the left side. The patient, a man aged about 45, had been for a year and a half under observation in hospital. He had been a law clerk, but was not over-worked; never had syphilis, nor had he taken mercury. About two years ago, (in October, 1849,) he was rather suddenly seized with dyspnœa and pains, which were of a rheumatic character.

"In June, 1850, when Dr. Mayne first saw him, he was complaining of severe cough, of a peculiar hoarse, raucous character, which gave the idea that the man was suffering from disease of the larynx, but, on examination, no other symptom of laryngeal disease could be detected. The voice was natural. He also complained of pain of a neuralgic character in the left shoulder, shooting to the acromion and along the superior intercostal spaces; it was intermittent, but never completely disappeared. In addition, he had dyspnœa, which, scarcely perceptible when he was tranquil, became distressing whenever he was hurried or ascended a height. He was sensible of a continued obstruction about the windpipe, and pointed to the upper part of the sternum as the seat of the affection. The pulse was similar in strength and character in both of the radial, the brachial, and carotid arteries. There was no turgescence of the jugular or of the other veins in the neck.

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[1] The patient, after leaving the Meath Hospital, came under Dr. Greene's care, who has recorded the whole case. See his memoir, already quoted, Dublin Journal of Medical Science, vol. vii. p. 237.

[2] Transactions of the Pathological Society, Dublin Journal of Medical Science, vol. xv. p. 154.

"The action of the heart was regular, and without any abnormal sound. At the upper third of the sternum there was dulness, which extended over the costal cartilages on either side for about two square inches; there was no *souffle* here, but two sounds were heard as in the heart, and a little later than these in point of time. There was an impulse, which was single, but stronger than that of the heart; both impulse and sounds diminished as the stethoscope was moved towards the heart, and again increased in the true præcordial region. Both sides of the chest were equally resonant on percussion, but the respiration in the upper part of the left lung was more or less bronchial both in front and posteriorly, and at the base of the lung it was not at all so clear as the resonance on percussion would have led him to expect. After some months the tumour in the upper sternal region became much more decided; it was visibly pulsatile, and the seat of a constant dull pain. The pulsation was diastolic. The note taken in November of the same year was, that the pulse and double sound continued, and that there was no *souffle*.

"He was again carefully examined in April, 1851, when it was found that the left side of the chest had become smaller than the right; the ribs seemed crowded together, and the whole side contracted, very like what takes place after the absorption of a pleuritic effusion. The contraction continued to increase. In August the affected side was less than the opposite by two inches; and shortly before death the difference had increased to three inches. In October it was noticed that the left side had also become contracted in the vertical direction; the shoulder had advanced, and the angle of the scapula had started from the side. The right side was preternaturally clear on percussion; the left was also clear, but there was a slight shade of difference between the two in this respect. In addition there was no true respiratory murmur to be heard in the left lung, except at its most inferior portion; at the upper part, both in front and posteriorly, it was bronchial or tracheal, and there was well-marked resonance of the voice. The heart was now displaced towards the left axilla. There were, in fact, all the appearances usually observed after the absorption of a large pleuritic effusion of the left side; yet of this disease he had none of the general symptoms, neither had he those of cirrhosis of the lung. The conclusion, therefore, arrived at was, that the left bronchus was compressed by the aneurismal tumour.

"There was never any hemorrhage, but the tumour gradually approached the surface, and the integuments became thin and discoloured, and sloughing seemed at hand. He died, however, without the sac having given way either externally or internally.

"*Autopsy.*—The aneurism was found to have arisen from the ascending portion of the thoracic aorta, before the giving off of any of the great vessels; it was double the size of an orange, and had caused absorption of the sternum, and of portions of the costal cartilages, upon either side. It had extended downwards towards the left lung, compressing and flattening the left bronchial tube. There was a slight and recent pleuritic effusion on the surface of the left lung. The mucous membrane of the œsophagus was ulcerated in two or three spots. There had been no dysphagia; but towards the close of the case the man had complained of pain in swallowing solids. The aortic valves were healthy."

We have next to examine the condition of the voice. Alteration of voice is by no means constant, and may be absent even when bronchial and tracheal stridor exist, a fact of great importance in the diagnosis between laryngeal disease and aneurism. In the former affection some alteration of the voice is almost always present, and may exist even without stridor. Stridor from laryngeal disease is always attended with lesion of voice; but in aneurism aphonia *without the stridor from below,* as in Dr. Smith's case must be very rare. It often happens that stridor occurs without aphonia.

In most forms of chronic laryngeal disease there is not only stridor, but a gradual loss of voice, passing through different forms of hoarseness, until aphonia is produced. When this is established it is generally constant, and in many cases the voice is never restored. In aneurism the reverse of this is observed: and we find remarkable variations in the tone and power of the voice occurring within short spaces of time. This is what might be expected from considering the cause of the symptom, which is manifestly the result of pressure, or of irritation, or of both combined, upon the recurrent nerve. In the case of aneurism of the innominata, already spoken of, the variations of voice were most remarkable, and occurred within short spaces of time. Within twenty-four hours it would change from the highest treble to a deep bass. At one time it was an inaudible whisper, at another hoarse and croaking; and this variability of voice continued up to the period of death. The recurrent nerve was found stretched over the tumour like a broad ribbon, and its fibres were evidently vascular.[1]

Let us contrast aneurism and laryngeal disease with reference to the alterations of voice, and the stridor.

Aneurism of the Aorta.	*Chronic disease of the Larynx.*
1. Lesion of voice frequently absent.	1. Alteration of voice generally present.
2. Occurrence of stridor without aphonia.	2. Stridor combined with aphonia
3. Alterations in voice variable.	3. Loss of voice much more constant and progressive.

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[1] It is still to be determined how far the aphonia in aneurismal disease is functional, or proceeds from some permanent change in the larynx. In some cases mucous inflammation is produced partly by pressure; and authors have spoken of the existence of œdema glottidis. I believe that in most cases the aphonia is originally functional, and that if organic change follows, it is likely to be that which would result from deficient nervous action. Dr. Todd has shown that, in a case where the recurrent nerve was flattened and compressed, atrophy of the vocal muscles, on the corresponding side, had occurred. The same observation has been lately made by Professor Banks. (See Transactions of the Pathological Society of Dublin.)

A similar condition of the larynx of a horse, in a case of "roaring," was lately exhibited by Dr. Smith to the Pathological Society of Dublin.

4. Stridor, in most cases from below.	4. Stridor, in all cases from above.
5. Respiration often feeble in one lung, and loud in the other.	5. Respiration (supposing the lungs to be healthy) equal in both lungs.
6. Stridor, with loud respiration in one lung.	6. Stridor, with equally diminished vesicular murmur in both lungs.
7. Stridor, with dulness on percussion, of the upper portion of one lung.	7. Stridor, without dulness (*the lungs being healthy.*)

Pressure on arteries and veins.—The arteries whose compression is of importance in diagnosis are the subclavian and common carotids. Other branches may, doubtless, be engaged, but these are the vessels most often affected; and the pressure is indicated, in the majority of cases, only by deficient or absent pulsation in the vessel or some portion of it distal to the seat of pressure. The failure of the pulse at one wrist, or a great inequality of the radial pulses, has long been recognised. From the naturally greater volume of the right than the left pulse, the sign in question has more value when occurring in the right than in the left radial artery. We have never met with a case of mortification of the arm from want of arterial supply. The pressure is exercised in a gradual manner, and time is given to establish a collateral circulation.

But with obliteration of the carotid physicians are not so familiar. It is more common than is generally supposed, and is probably the cause of the cerebral disease which not unfrequently complicates aneurism of the thoracic aorta and of the innominata.

Here the lesion of the brain is probably of that kind indicated by Dr. Law, which proceeds from deficient arterial supply. In a case of obliteration of the carotid, attendant on aneurism of the innominata, the period of commencement of the aneurismal disease was easily determined; and from this time up to that of the occurrence of hemiplegia of the left side, the patient suffered from pain in the head, vertigo, tinnitus aurium, flashes of light in the eyes, and occasional numbness of the left arm and leg. The paralysis, indeed, occurred in the course of one night, but this is consistent with the view which has been taken of its nature.[1]

So far as the actual condition of the arteries under pressure is concerned, it may be one of obliteration of the tube for some extent, the artery degenerating into a fibrous and flattened cord, or there may be beyond the point of pressure a healthy and patent condition of the vessel. In certain cases pressure may exist, even for a considerable time, and yet no obliteration at the point of pressure take place; for it has happened to me more than once to observe the return of the radial and tracheal pulses in a case where they had been absent. This is one of the indications of the curious circumstance presently to be described, namely, the occasional change in the direction of the tumour. It is probable that, in some instances, a process of arteritis may be set up in the vessel which is compressed. Thus, in a case where both the carotid and subclavian arteries were pulseless, we found the carotid obliterated as far as it was traced, and converted into a hard cord, while the subclavian, immediately beyond the point of pressure was gaping, and perfectly healthy.

Compression of the nutrient arteries of the Lung.—It has been shown by Dr. Carswell, that pulmonary gangrene is induced by compression of the nutrient arteries of the lung:[2] the sequence of phenomena is as follows:—

1. Signs and symptoms of intrathoracic tumour.
2. Symptoms of gangrene of the lung.

In a case by Dr. Greene[3] of compression of the left bronchial tube by an aneurism, two remarkable symptoms were developed some days before death: one, an emphysematous swelling on the front of the chest, extending down both arms, and alternately affecting the abdomen, back, and scrotum; and the other, the expectoration of an extremely fetid matter. The patient died, with symptoms of progressive exhaustion. The left bronchial tube was found greatly compressed and in a state of slough. The sloughing process had extended to the trachea and larynx. In the upper portion of the lung existed some small gangrenous cavities, and the lower lobe was more or less sphacelated (the uncircumscribed gangrene of Laennec.)

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[1] Dublin Journal of Medical Science, vol. v. p. 406. See also Dr. Law's Paper on the Connexion between Cerebral and Cardiac Diseases, in the same Journal, vol. ix.

[2] See his Pathological Anatomy.

[3] Transactions of the Pathological Society of Dublin, Dublin Journal of Medical Science, vol. xvii. p. 522. This case was observed in 1836, and in it also double impulse is recorded.

This case, taken in connexion with the facts described by Dr. Carswell, is an important addition to our knowledge, not only of the diagnosis of intrathoracic tumours, but of gangrene of the lung. In the latter affection there is nothing more remarkable than the frequent want of accordance between the severity of the symptoms and the amount of physical evidence of disease, the latter being often absent even for a great length of time.[1] In such cases the disease may be the diffluent gangrene of the bronchial membrane described by Rokitansky.[2] I have never met with mortification of the lung consequent on aneurism, but have described a case of this result in soft cancerous tumour of the posterior mediastinum, which simulated an aneurism of the aorta. The left bronchus was compressed, and respiration was generally feeble in the left lung. Some days before death, the patient suddenly expectorated a great quantity of fetid sanies and purulent matter. A large encephaloid tumour was found springing from the posterior mediastinum; it surrounded the left branch of the pulmonary artery, which was flattened and narrowed. The lung was pushed from above downwards; and in its upper portion there existed a gangrenous cavity of the size of a pullet's egg.[3]

In this case we had diastolic pulsation, double sounds, systolic bellows murmur, dulness on percussion, dysphagia, tracheal stridor, inequality of respiration, and feebleness of the left radial pulse. The voice was unaffected. In addition, the patient had convulsions, paralysis, pain of the shoulder and side, and hæmoptysis. I believe that this is the first instance in which the signs of pulsating intrathoracic cancer were observed. Mr. Carmichael with whom I saw the patient, thought that the case was one of aneurism. I only ventured on the diagnosis of tumour for two reasons; one, that the symptoms had continued for upwards of four years; and the other, that although the dulness was well marked and extensive, the diastolic pulsation was much feebler than we might expect from a large aneurism in contact with the ribs.

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[1] Clinical Researches on Gangrene of the Lung. Dublin Quarterly Journal of Medical Science. No. xvii. 1850.

[2] See his Pathological Anatomy, vol. i.

[3] Researches on the Pathology and Diagnosis of Cancer of the Lung and Mediastinum, Dublin Journal of Medical Science, vol. xxi. p. 227.

The frequent absence of physical signs in gangrene of the lung being borne in mind, and also the fact that, in Dr. Greene's case and in that now given, signs of compression of the bronchial tube preceded the fetid expectoration,—we may conclude, that where the latter symptom has been preceded by feebleness of respiration in one lung, the combination of pulmonary gangrene with an intrathoracic tumour probably exists.

Pressure on the Veins.—As an indication of intrathoracic tumour, an extensively varicose state of the superficial veins of the neck and thorax is probably less frequent in aneurismal than in cancerous disease. The pressure may be exercised on the venæ innominatæ or the superior cava, and is shown by enlargement and tortuosity of one or both jugular veins, and of the superficial veins of the thorax. The superior cava may be adherent to the tumour, and become narrowed, not only by pressure, but by adhesion of its internal surfaces.

This condition existed in a case of varicose aneurism observed by Dr. Mayne, which we shall give in the appendix to this chapter.

In other cases we find that in place of the large tortuous veins ramifying on the surface, there is a puffy, elastic swelling of the entire neck. To this may be given the name of the tippet-like swelling of the neck. It is very characteristic, but by no means constant.

The dilatation of veins, from the pressure of an aneurismal tumour, differs from that observed in diseases of the heart in some particulars. It is sometimes confined to, or it greatly predominates at one side. The mammary and intercostal branches are more often manifest; and the regurgitant pulsation has not, so far as I know, been observed. The undulations caused by the lateral impulse of the carotid or subclavian may be seen. We have not met with the tippet-like swelling in cases of disease of the heart.

Finally, it may be stated, that a varicose condition of the veins which supply the superior cava, occurring in a case where no signs of mitral contraction, with its attendant pulmonary congestion and dilatation of the right cavities, or of other disease of the heart, are presented, should point to the diagnosis of an intrathoracic tumour.

Compression of the Œsophagus.—This condition, though not uncommon, is less frequently observed than compression of the air-passages. According to Dr. Greene, it is most often met with when the aneurism springs from the transverse and descending portion of the arch than elsewhere. We have not found it in aneurisms of the ascending aorta; but it may be present when the disease is in the innominata or the lower portion of the descending aorta. It may exist with or without stridor, and in some cases is almost the only symptom complained of. In such cases the aneurism is probably a very small one, and situated low down in the course of the vessel. In a case already alluded to,[1] the patient, when admitted into my wards, made no complaint but of the dysphagia, which was attended with eructations and loss of appetite. Ten days previously he had been attacked with pain in the back, and stitches referred to the lower portion of the sternum, where he also experienced a dragging sensation. No physical sign of intrathoracic disease existed, except feebleness of the vesicular murmur in the right lung. The aneurism, which was not larger than a pullet's egg, burst into the œsophagus, which it had compressed against the right bronchus.

Aneurismal dysphagia exhibits a certain variety in its symptoms. It is commonly referred to the middle third of the sternum, but may be felt lower down. And in the same case, as has been shown by Dr. Law, it may be referred, successively, to different portions of the tube,—now at the top of the sternum, and again to the epigastrium.[2] The difficulty of deglutition varies from complete dysphagia, which is rare, to the slightest feeling of obstruction; generally fluids are most easily swallowed. The sensation may be merely one of pain without that of mechanical obstruction. The attempt to swallow often produces a paroxysm, compounded of retching, convulsive hiccough, laryngeal cough, and dyspnœa. Pain, and a sense of weight, followed by hiccough and vomiting, or, rather, regurgitation on the attempt to swallow solids, is frequent. A copious draught of fluid, when the mass of food remains above the stricture, sometimes causes it to pass down; and, under these circumstances, auscultation between the shoulders enables us to hear, during the passage of the food and drink through the narrowed tube, a series of singular and characteristic sounds. Sometimes, the taking even of a mouthful of fluid produces a sort of spasm, and for a time the ingesta cannot be passed either upwards or downwards; and this state is attended with dyspnœa or arrest of breathing.

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[1] Cases of Internal Aneurism. By William Henry Porter, M. D. &c., Dublin Journal of Medical Science, vol. iv.

[2] Transactions of the Pathological Society of Dublin, February 27, 1841.

In a case observed by Dr. Law, the patient could not swallow in the recumbent position, but always took his food while sitting up, and with the body bent forwards, and to one side. The aneurism was multilocular, and of great size, lying on both sides of the spine. It is yet to be determined how far the use of the probang will assist in the diagnosis between this form of dysphagia and organic stricture of the œsophagus. Of course, if an aneurism be suspected, exploration by the probang will hardly be resorted to, especially as cases have occurred where the aneurism has ruptured under the operation. But when we remember, that aneurismal dysphagia may arise and disappear several times in the course of the case, and with intermissions even of long duration, it appears probable that the œsophagus can bear the pressure of a tumour for a long period without disorganization, so that there may be periods when the passing of the probang, if not justifiable, might be at least safe. It is also probable that, in cases of rupture under the operation, the cause of the accident was rather the convulsive spasm of the parts than the mere action of the probang in lacerating the coverings of the tumour. In my case, as recorded by Mr. Porter, the probang was passed without any decided obstruction being felt. The operator was sensible of the instrument passing over a soft tumour. In this case the hemorrhage did not occur until the seventh day after the operation. In a case by Dr. Law, the passage of the probang was arrested at a point about four inches below the isthmus faucium; no injury followed; and the patient lived for four months afterwards, and did not die by rupture of the sac.

It is highly improbable, however, that, by the use of the probang, we shall ever be enabled to distinguish between aneurismal strictures and those which proceed from soft cancers, or of scrofulous tubercle under the mucous membrane. As a general rule the instrument should not be used until the patient has been carefully examined for the existence of stridor from below, or the inequality of respiration in the lungs.

In connexion with the symptom of dysphagia, the following case possesses great interest, as an instance of long-continued intermissions of the obstruction, and of ultimate perforation of the tube, yet without hemorrhage.

CASE LXXII.—*Aneurism of the Arch of the Aorta, with perforation of the Œsophagus and left Bronchial Tube; Dysphagia and stridor from below, both presenting long intermissions; Disappearance of the physical signs of Aneurism; Death without hemorrhage.*

A gentleman, aged 39, was attacked in May, 1841, with pain in the lower portion of the back and sides. It came on rather suddenly, and ultimately fixed at about the eighth dorsal vertebra. Under the supposition that the disease was caries of the spine, caustic issues were inserted on each side, and as soon as purulent discharge was established, all the symptoms were ameliorated. In a few months, however, cough, with a crowing sound, came on, attended with dyspnœa, which was aggravated by exertion, or by any attempt to stand unsupported. So long as the patient remained on crutches, or sat bent forward, his symptoms were relieved. Dysphagia set in. At this time I first saw the patient, in consultation with Sir Philip Crampton and Dr. Smyly. A bellows murmur, loudest at the upper and anterior portion of the left side, had been observed by the latter gentleman.

We now found that the breathing was stridulous from below, the left pulse feeble and small, while the right was large. There was dulness of the left clavicle and supero-anterior portion of the side, and here there existed a strong diastolic throb, attended with bellows murmur. Of the existence of a tumour there could be no doubt, and we agreed that it was probably an aneurism.

Within a few months all these physical signs began to disappear. The dysphagia ceased; the bellows murmur was no longer heard; the pulse at the left wrist was restored to its natural character; and the patient continued in good health for more than a year, except whenever the discharge from the issues was suspended or diminished. Early in 1843, the use of the issues having been omitted, the dysphagia returned with greater severity than on any former occasion. He was brought to town, almost dying from inanition, when Dr. Smyly re-opened the issues, and again he was relieved from the dysphagia, and his general health and appearance restored. At this time I made a careful examination, but could detect no physical sign of aneurism, or of any tumour within the thorax. On the 15th of April, 1843, he was attacked with diphtheritis, followed by bronchial effusion and death. There was no hemorrhage.

On dissection the heart was found healthy. At the commencement of the descending aorta existed a large false aneurism, filled with coagula, and showing on its posterior surface the bodies of five dorsal vertebræ, deeply eroded. The left bronchus and the œsophagus were both strongly compressed by this tumour. In the first existed a perforation, communicating with the sac, and closed by a coagulum. In the œsophagus were two perforations, nearly opposite, and, as it were, passing across the compressed portion of the tube. These also were closed by coagula. The aorta was thickened and atheromatous, and in the apices of both lungs were found calcareous matter and old tubercle.

This case is deserving of study, but our space only permits us to enumerate its more important features. They are;—

1. The disappearance of many of the symptoms and of the usual signs of aneurism.

2. The evidences of a change in the seat of the tumour, and the relief of surrounding parts from pressure.

3. The relief obtained from the use of crutches, which must have acted by removing pressure on the tumour, caused by the bending downwards and forwards of the spine, in the seat of the erosion.

4. The singular fact, that almost all the symptoms seemed to be suspended just in proportion as the discharge from the issues was promoted. Even the dysphagia, after it had existed to such a degree as that death from inanition was threatened, disappeared on the re-opening of the issues.

The complete cessation of the sounds and murmur of the aneurism, and the evident retreat of the tumour from the anterior portion of the thorax, can only be explained by supposing that a new chamber for the disease had been formed at the expense of the bodies of the dorsal vertebræ.

Combinations of Aneurism of the Thoracic Aorta.—We may divide cases of these combinations into two classes, viz., those with diseases which accompany the lesion, but are not produced by it, and those in which various organic or functional lesions are consequent upon it.

As might be expected from thoracic aneurism being so often met with in men of strong constitutions, the class of associated or preceding diseases is but limited; and there are no local affections of organs distinct from the aorta which appear to have any frequent association with aneurism. The morbid condition which most often accompanies aneurism is that of tubercle; and yet, under these circumstances, the symptoms of phthisis are often doubtful, irregular, and slowly progressive. This is what might be expected, when we recollect the period of life at which aneurism is commonly developed. The combination is more frequent than has been supposed, and, in some instances, death occurs without rupture of the aneurism, the symptoms being those of ordinary phthisis. I believe that in most instances, where the symptoms of both diseases are manifest, the arterial lesion has had the initiative; and I have often thought that there was a case deserving the name of consumptive or strumous aneurism, in which the same general morbid state which caused deposition of tubercle in the lung simultaneously affected the coats of the aorta.

Among the associated or consequent diseases we may enumerate:—

1. Hypertrophy of the heart.
2. Gangrene of the lung.
3. Paralysis, probably from arterial obstruction.

With reference to the first, we may hold that its occurrence is accidental. In some instances hypertrophy has preceded the aneurism, or there has been a deficient state of the aortic valves; but where the heart or its valves have not been previously engaged, there is no reason to believe that the existence of aneurism in any portion of the aorta throws additional labour on the heart; and hence we commonly find a healthy heart co-existing with a vast aneurism. And this leads to the conclusion, that the force of the aneurismal pulsations does not merely depend on that of the ventricular systole. I showed in 1834 that the explanation of the violent shock of an aneurism was to be found by referring to the laws of hydrostatics, and that, as in Bramah's press the injection of a small quantity of fluid into a reservoir gives a force greatly superior to that by which it was introduced,—so even a feeble systole of the ventricle may cause a powerful diastole in an aneurismal sac. The most violently pulsating aneurism which I ever met with co-existed with a small and atrophied heart. The aneurism was of great size; the sac stretching downwards into the abdomen; and the sufferings of this patient, from the mere throbbing of the tumour, were indescribable. When he lay down, he felt as if the pulsations would throw him out of bed. The pulsation will, of course, be greater in proportion to the fluidity of the contents of the aneurism. In a false aneurism, with but little coagulum, and with a form more or less globular, we can easily understand how the surrounding parts will be torn up and destroyed by this tremendous action.

Mode of Death in Aneurism.—An aneurism having been found to exist, it is impossible to predict what direction it will take, what will be the duration of life, or by what mode the patient will die; nay, we cannot even affirm that the patient will die of the disease at all. His death may take place from totally different diseases, either acute or chronic, more especially if the progress of the aneurism has been delayed or arrested by judicious treatment. In persons who are free from disease of the heart, in whom, too, the aneurism does not compress any important organ, such as the trachea or œsophagus, death, without rupture of the aneurism, may, in fact, occur in various modes. The patient may sink from the effects of pressure on surrounding parts, or be worn out by pain, sleeplessness, and irritative fever; he may die of some accidental acute disease, such as pneumonia, cholera, or fever, or sink with the symptoms of phthisis, gangrene of the lung, or disease of the brain.

We have already seen that perforation of hollow organs does not necessarily imply hemorrhage, and that although aneurism has formed a communication between the left bronchial tube, the trachea, and the œsophagus, yet that death may occur as if there had been no aneurism at all. This seems more likely to happen in chronic cases in which the whole quantity of circulating blood has been diminished, and where the aneurismal cavity itself is at last imperfectly filled. Under these circumstances, its expansive force will be lessened, and the coagula which block up the perforations remain undisturbed. Again, it may happen that death will not be immediate, even where a copious and sudden hemorrhage has occurred.

This may be observed not only when the aneurism opens internally, but also when it bursts through the integuments. The following case occurred some years ago in Sir Patrick Dun's Hospital, under the care of Dr. Osborne.

A woman, aged between 35 and 40, was received into the hospital with a pulsating tumour, evidently aneurismal, which presented in the lower sternal region, and appeared to have caused absorption of the bone. After some time a well-defined circular discoloration of the skin, the size of a halfpenny, took place exactly over the most prominent part of the tumour. This was soon followed by a slough and separation of the integuments and subjacent structures, so as to display what appeared to be the outer surface of the coagulum of the aneurism. At each systole of the heart this coagulum moved outwards, so as to close up the orifice in the skin, and then receded during the diastole. The action thus produced was aptly compared by Dr. Montgomery to that of the valve of a flute when closing the opening. This patient was of a lively and talkative disposition, and suffered so little that it was with difficulty she could be prevented from singing aloud. After some days the coagulum gave way, and a deluge of blood was poured out. Immediate death would have followed but for the presence of mind of the nurse, who, on the instant, snatched up a cotton apron, and stuffed a portion of it into the opening of the chest. The patient recovered for the time, and for many days subsequently exhibited the extraordinary spectacle of a person in high health and spirits, whose life was depending upon the precarious support of a plug of cotton rag, which at every stroke of the heart appeared on the point of being forced out of its situation. The result was, of course, fatal.

In the Museum of Anatomy of the University of Dublin, Professor Harrison has a cast, exhibiting the appearances in an aneurism where the disease proved fatal by frequent losses of blood through an external opening. The particulars of another case have been given to me by Professor Smith.

A woman, aged 46, presented a large aneurism of the thoracic aorta, which had perforated the sternum, and formed an external tumour, covered merely by integument. The latter was slightly discoloured, and obviously becoming thinned. Shortly after her admission into the Richmond Hospital, the integuments gave way, and an opening, as large as a fourpenny-piece, was formed, through which a large quantity of blood, partly fluid and partly in clots, suddenly issued. This bleeding was, in some measure, checked by a large coagulum blocking up the orifice, and was for the time arrested by the compress and roller. A slight oozing continued for a day or two, when another alarming hemorrhage took place, which was again commanded by the use of additional compresses. In this way the case proceeded for about ten days, the blood gushing at every second or third day, and oozing out in the intervals. The patient died from exhaustion, but not by any sudden syncope. On removing the compresses, the external opening was found greatly enlarged, being of the size of a half-crown piece.

The following case, for which I am indebted to Dr. Neligan, may be studied with advantage, especially with reference to the views of Gairdner as to the nature of certain cases of hæmatemesis, in which he holds that the bleeding results from the leaking of an aneurismal tumour.[1]

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[1] Edinburgh Monthly Journal of Medicine, Third Series, vol. i. 1850.

CASE LXXIII.—*Pulsating tumour in the region of the Arch of the Aorta; Absorption of the Ribs, and perforation of the Integuments; Frequent Hemorrhages, and signs of the diminution of the Tumour; Great improvement of the health under a generous diet.*

A ship-carpenter, aged 56, was admitted into Jervis-street Hospital in November, 1844. For three years he had suffered from a severe cough and paroxysms of dyspnœa, which he ascribed to the presence of a tumour within his chest, existing between the second and the fifth ribs, on the right side anteriorly. Its form was oval, and its long diameter from above downwards. Over this tumour the sound was dull on percussion; a strong pulsation, synchronous with the heart's systole, was to be felt; and auscultation detected a loud, double bellows murmur. The integument had a somewhat darker colour than that of the neighbouring skin; but the third and fourth ribs seemed to have undergone absorption towards their sternal extremities, and where they passed over the tumour. The right pulse was considerably larger than the left, and the patient suffered from a severe cough occurring in paroxysms. His voice was peculiar and husky.

At the upper edge of the tumour was a red spot, of about an inch in diameter, in the centre of which existed a small valvular orifice; from this, at each pulsation of the heart, bloody serum was discharged in jets. The opening appeared to be sinuous, and to have a communication with the tumour.

The patient stated, that for the last year blood had been discharged from this opening at various periods. The hemorrhage was sometimes very copious, and occurred in a continued stream. He mentioned that at first the skin covering this spot became gradually thinner, until at length a large *blob of blood* appeared, which burst and bled profusely. The bleeding was arrested with great difficulty, but for some days a bloody fluid continued to escape from the orifice. The same process was gone through every time that the tumour opened.

It appeared that after having suffered for some months from attacks of palpitation, which used to occur twice in the twenty-four hours, and were attended with severe pain in the lower part of the right side, gradually ascending to the site of the tumour, he perceived the ribs to be bulged out, when the symptoms now complained of set in and gradually increased in severity. Two years before, he had been treated by repeated bleedings and a low diet, from which he thought he derived benefit. Since then, however, the tumour had increased in size.

In about a week after his admission into Jervis-street Hospital the cuticle covering the sinuous opening had become more and more distended, until it formed a bloody tumour of the size of a large nut. On the evening of the 3rd of December this tumour burst, and a small stream of blood continued to issue from it in jets. Notwithstanding the use of firm pressure, a large quantity of blood was thus lost, until the patient became faint, when the bleeding ceased, on the application of dry lint and the use of continued pressure with the hand.

On the 31st of December the tumour was found to have considerably diminished in size. It now communicated the sensation of impulse produced by a much more solid substance than at first, the soft, fluctuating feel being completely changed. In about a week another hemorrhage took place, but the quantity lost was much less than before. He left the hospital on the 1st of February, 1845, stating that he felt quite well. His cough and dyspnœa, had nearly disappeared. The tumour was still manifest, it felt firm, and gave the sensation of a solid pulsating mass. The sinuous opening had completely healed over, and the discharge of bloody serum had ceased.

The treatment pursued in this case was the exhibition of small and repeated doses of morphia, with the use of a nourishing diet, in moderate quantities. The pulse, which had been 76 on admission, fell to 48, at which rate it steadily remained.

That this singular case was one of the external opening of a large aneurism of the aorta is more than probable. The early history, and the physical signs, all indicate this disease. The opening was probably small, and the passage of the blood took place by a very sinuous canal, and in this way the sudden gush of blood in any quantity was prevented.

The only question that might be raised is, whether the disease was a fungous tumour, but the following considerations should make us reject this view of the case:—

1. That the early symptoms were those of aneurism in a well-marked form.

2. That the pulsations of the tumour were strong, and *with a double murmur.*

3. That the external tumour had no character of fungous or malignant structure, and that there was no appearance of carcinomatous disease on the surface.

4. That the patient's health was in a comparatively good state at the end of the third year, although at one time it had greatly given way. This could hardly have occurred in malignant disease.

We have observed two cases in which successive hemorrhages in quantity took place from an aortic aneurism. In each case the opening of the sac was into the right pleura. The symptoms and signs were very similar in both, each gush of blood being attended with collapse, and producing a dulness of the lower portions of the side, exactly corresponding to the amount and situation of the effused blood. In one of these cases there were two, in the other three, distinct hemorrhages; and in the latter the state of the patient, and the condition of the pulse, showed that no bleeding was going on during the intervals. In this instance the patient had been cured of a popliteal aneurism. After some months he was attacked with pain in the lower part of the left side of the chest, and the existence of an aneurism, probably in the descending thoracic aorta, was suspected. The disease in the course of a few months became but too plain. I saw this patient soon after the first hemorrhage, which was attended with a violent fit of convulsions and disappearance of the pulse. The lower portion of the right side had become dull for about a hand's breadth. He soon rallied, and the circulation was restored. In a few days a second attack, with precisely the same symptoms, took place. The dulness was now found to have ascended beyond the apex of the scapula. The third hemorrhage was fatal, and it occurred in my presence. Immediately before the attack the patient appeared perfectly well. He was cheerful, had a good colour, a full, strong, and regular pulse; his appetite was good, and respiration tranquil. He had occasion to turn on the left side, when, in an instant, a fearful attack of convulsions came on. The pulse disappeared, and after a few spasmodic respirations he was dead. On dissection, the whole sac of the pleura was found filled with blood, which had separated into crassamentum and serum. The aneurism had ruptured at the lowest portion of the thoracic aorta. We were unable to discover anything that could explain why the first and second hemorrhages had been arrested. The heart was healthy.

But this sudden and stormy death in aneurism is less often met with than many might suppose; existence being frequently terminated by a slower process, sometimes attended with but little pain, at others by protracted suffering. Again, there may be sudden death without pain or distress of any kind, and without rupture of the sac. Of this several instances have been observed in Dublin. I have myself met with one in a case of false aneurism; and in the Richmond Hospital collection are specimens of true aneurisms, in which the death, though sudden, was unexplained by dissection. Such cases, however, are exceptional. In most instances of sudden death we observe syncope, with or without convulsions or asphyxia. The occurrence of either of these conditions depends, in a great measure, on the direction of the hemorrhage. If the aneurism burst into a serous sac, or into a free canal, such as the œsophagus, fatal syncope may be the result; while death by asphyxia is produced when it opens into the air-passages, or into the substance of the lung itself, which may be at once and extensively lacerated by the torrent of blood.

Yet death by internal hemorrhage is not always sudden, nor attended by distinct attacks of syncope. And this is true even when the blood is poured into a serous cavity. Of this an instance was lately observed in Dublin; the opening was into the pericardium, and yet, owing to partial adhesions, probably of long standing, the dilatation of the sac was gradual, and the sinking of the vital powers very protracted.

We have seen that sudden death may occur without rupture of the sac; so also may gradual death be caused from the effect of the aneurism on surrounding parts. There may be coma from compression of the veins, or slow disease of the brain from obstruction of the carotids. Mortification of the lung may result from pressure on the bronchial arteries, or the patient die of inanition, as when the œsophagus is compressed; or of asphyxia, in the case of a prolonged pressure on the windpipe, with increasing stridor. I have witnessed cases of the latter kind which make me believe that of all modes of death this is the most terrible. Looking back on these cases, the mind shrinks from contemplating the agonies which for weeks together the sufferer has had to endure.[1]

The last mode of death which has come under my observation is that in which the patient sinks, worn out by the sufferings attendant on the disease. In such cases the sac is generally a very large one. In addition to the pains and distress from the throbbing of the tumour, which often prevent sleep, the patient falls first into a state of cachectic anæmia, and next into one of irritative fever. The most remarkable instance of this kind I ever witnessed occurred in a patient who had been reduced immoderately by the vain attempt at cure by the use of the method of

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[1] To explain the different degrees of suffering to which, with apparently the same diseases, many patients are liable, is a difficult task. Yet the experienced physician must admit not only that in most cases the act of dying is painless, but that even long before the final struggle is encountered the condition of the patient is more terrible to the bystanders than to the sufferer. Putting aside instances of coma, febrile delirium, and a large number of the convulsive diseases, in all of which the patient himself is unconscious of pain, even though, as in some cases of tetanus, the countenance expresses agony the most intense, we shall meet examples where full consciousness is preserved, and yet the suffering be more apparent than real. Even the extreme of stridor may exist, and yet the patient declare that his breathing is easy. It is now many years since I attended a professional brother, who, after having passed middle age, became the subject of phthisis in its usual form. A large cavity existed in the left lung, but the trachea and larynx remained unaffected. He was attacked during the night but one preceding his death with all the symptoms of tracheal or laryngeal obstruction, and so great was his distress that the immediate performance of tracheotomy was discussed at a full consultation, summoned after midnight. For two reasons it was determined not to attempt the operation; one, the previous history of the patient, and the other, that the excess of stridor seemed owing to the sudden plugging up of the left bronchus. During the night and part of the next day he remained in a state of dreadful orthopnœa, while the stridulous respirations were so marked and prolonged that the patient might be described as breathing through a pin-hole. While supporting him in bed, I said, "M., I grieve to see you in such suffering." He replied, in the lowest whisper, "I have no suffering; I never was more free from suffering." Yet each inspiration was prolonged to at least ten times its usual duration. I said, "But your breathing is so difficult." He pressed my hand, saying, "Thank God, I never was breathing better." Next day the terrible stridor subsided, his voice returned, and for many hours before his death, quiet of body and of mind was mercifully given to him.

Valsalva. The aneurism was a vast one, engaging both the descending thoracic and abdominal aorta.

Before entering on the treatment of thoracic aneurism we shall lay down a few rules to assist the practitioner in his examination of a case of suspected aneurism. These will principally apply to cases in which no external tumour has appeared.

Aneurism should be suspected in cases where we find certain well-marked forms of thoracic suffering to co-exist with an unimpaired state of the general health. Of these sufferings the most important are pain and the occurrence of dyspnœa on exertion, both of which have recently supervened in an individual who has no sign of disease of the lungs or heart, and whose past and present state of general health has been good.

The history of the patient having been accurately taken, the observer should first proceed to examine the heart. If he finds its action regular, its sounds free from murmur, while percussion shows no enlargement, he may, for all practical purposes, assume that the organ is healthy. Under these circumstances, should an aneurism really exist, its discovery will be comparatively easy, unless in some exceptional cases.

The state of the lungs and liver having been, as far as possible, determined, the next step will be to examine by the eye for the existence of any pulsation distinct from that of the heart. This is to be sought for in the upper portions of the chest, the front of which must be stripped, and observed first at a certain distance, and then, as Dr. Greene has advised, by bringing the eye to a level with or a little below one shoulder of the patient, so as to enable it to glance across the chest. The patient should be placed between the observer, and the light should fall as near as possible on the mesian line. In this way a localized pulsation of the upper sternal or subclavicular regions, which would escape a less accurate examination, may often be discovered.

But the value of this pulsation, as a sign of aneurism, depends on its localization, and on the absence of that condition of the arterial pulse which so commonly attends permanently open aortic orifice. It is true, that aneurism might co-exist with this lesion; but the combination is, I apprehend, not a common one. And we should be slow in diagnosing aneurism when the throbbing of the upper sternal region is repeated in the carotids and the rest of the arterial system.

The aneurismal throbbing, then, differs from that of the permanent patency of the aortic valves in being much more localized, and in not being attended with the visible and increased pulsation of the vessels of the neck.

Manual examination is next to be employed. The observer should apply one hand firmly and extended between the scapulæ, while the other is placed upon the upper portion of the sternum. Upon making pressure with both hands, a deep-seated diastolic throb, most evident at the end of expiration, may often be detected. The hand that was placed between the shoulders is now to be removed to the region of the heart, and the two impulses compared as to their force, character, and synchronism; and the observer may then find not only that the upper impulse is the stronger, impinging on a larger surface, and with a more extended diastolic throb, but that the two impulses do not exactly correspond in time; the cardiac is, of course, the first, and where a distinct interval of time between the pulsations cannot be found, we may still observe that the impulsive action commences from below. The slower the action of the heart, the more applicable will be this test, which I have verified even in a case of aneurism of the ascending aorta. I think that when the pulse is above 70, the difference in time can hardly be observed; and in such a case the use of digitalis might be had recourse to as a means of facilitating diagnosis by reducing the rate of the heart's action.

The evidences of pressure on surrounding parts are next to be sought for; but it must be recollected that an aneurism may exist without a single one of the phenomena which attend eccentric pressure. We must observe the condition of the veins of the neck, which, when they exhibit a varicose state at one side only, are strongly indicative of intrathoracic tumour. An enlarged condition of the veins on both sides, with or without the tippet-like swelling of the neck, unattended by the reflex pulsation, or the peculiar action of the heart which attends dilatation of the right cavities, furnishes a diagnostic of great value.

The next step will be the comparison of the radial pulses, it being borne in mind that in many persons the pulse in the right arm is larger than that in the left; and a careful comparison should also be instituted between the brachial, subclavian, and carotid arteries on both sides.

We are lastly to examine for the stridor from below, the alterations of voice, the character of the vesicular murmur in both lungs; and finally as to the occurrence of dysphagia. The character of variability in degree, and in the apparent point of pressure, belongs to all the symptoms in this class.

These investigations are preliminary to the use of percussion and the stethoscope as applied to the direct examination of the aneurism. If by careful percussion of the clavicles, scapular spines, or any part of the antero-superior portions of the chest, we find a localized dulness, in which situation there is either an unaltered vesicular murmur, a diminished murmur or a tracheal sound, and all without râle, we get an important result. In many cases the aneurismal dulness is most developed at a point below the clavicle, and in some an almost perfect pectoriloquism is communicated across the tumour.

Finally, the proper acoustic signs of aneurism are to be sought for; and the sounds, single and double, and with or without murmur, carefully compared with those of the heart. If, as we have already observed, the heart is healthy, and the aneurism free from murmur, we have then to compare the sounds of the tumour, single or double, with the double sounds of the heart. And in some cases we find that the second sound of the aneurism is louder and sharper than that of the heart. Again, if murmur exist in the aneurism, while the heart's sounds are free, we at once obtain an important diagnostic.

The conditions of the heart, however, may be various. We may find:—

1. The impulse and sounds of the heart natural.
2. Some increase of impulse, with a small pulse, and a murmur attending the first sound, having its greatest intensity in the situation of the mitral valve.
3. Murmur, with the first sound predominating at the base of the heart; the second sound healthy.
4. Double murmur replacing the second sound, audible at the base of the heart, and in the course of the substernal aorta; the pulse in this case will be probably large and throbbing.

It is obvious, that it is in the first two cases the discovery of a murmur, localized in some portion of the thoracic aorta, will have the greatest value as a diagnostic of aneurism, inasmuch as such a murmur cannot be supposed to proceed from the heart. In the first case there is no murmur to be communicated; and in the second, its seat being in the mitral and not the aortic valves, it is not propagated to the artery. An aneurism, doubtless, might exist in cases where either the third or fourth condition, now indicated, was present; but it is plain that under such circumstances the discovery of a murmur in the aorta would have a diminished value as an indication of aneurism. It would, in fact, have none unless it was found to possess a different character, and a greater degree of intensity, at a portion of the vessel more or less remote from its origin.

The stethoscope is now to be carried along and on both sides of the mesian line, extending laterally to a distance of about three inches on each side; and careful exploration should be made to discover a localized bellows murmur in these situations. We should also examine along the spine with the same view.

It has been already shown that in many cases of aneurism there is no murmur produced; and the physical signs are either altogether wanting, or consist of a single or double pulsation. I believe that the liability to double pulsation increases as we approach the heart. When murmur is present, it is commonly, as Dr. Hope has described, hoarse in sound and abrupt in character.

I am not prepared to say whether it is ever actually double, or whether in the cases of double pulsation there may not be an abortive second murmur.

TREATMENT OF ANEURISM OF THE THORACIC AORTA.

In discussing this question we must be content to deal with palliative rather than curative treatment. The efforts of the physician should be directed, on the one hand, to retard disease, and, on the other, to diminish suffering. It is to be doubted whether we are justified in adopting any measures which, while they are directed under theoretical views to the cure of the disease, materially interfere with the patient's condition. It often happens that a patient who has not been thus interfered with will continue with unimpaired health and strength for a great length of time until he is so unfortunate as to be placed under treatment for the cure of his aneurism. For then all the evils which have been pointed out as occurring in cases of indolent disease of the heart, when injured by ignorant treatment, are induced. The patient's mind becomes excited and apprehensive; his system is weakened by depletion; and his digestive functions ruined by starvation. The forces by which he can resist disease are broken down, his blood becomes uncoagulable, his tissues unresisting. The force of the aneurismal throb is augmented, and a disease which, under other circumstances, might have endured for years with but little interference with the general health, is turned into a rapid and destructive malady.

The practitioner should never forget that local diseases, themselves incurable, may nevertheless co-exist with an excellent state of the general health, for a period indefinitely long, and the conclusion is but too obvious, that as the lesion cannot be cured, the system at large should not be tampered with.

In a practical point of view, we may divide cases of aortic aneurism into two classes:—

1. Those in which the action of the heart is undisturbed, and in which there is no physical sign of disease in the valves or cavities of the organ. This case is not uncommon.

2. Cases in which we have the combination of thoracic aneurism with an excited and often an organically diseased heart.

We might, perhaps, add a third class, of which I have seen a few examples, where the force of the heart was below the healthy standard, with or without atrophy of the organ.

In the first of these cases, any medical interference, beyond what is necessary for the relief of pain is unjustifiable; in the second we may attempt to moderate the force of the heart; and in the third we are called on to follow the treatment proper in cases of weakened heart.

The so-called treatment of Valsalva has still some followers in these countries, inconsistent though it be with all that we know of pathological anatomy and practical medicine.

If, in a case of false aneurism of the aorta, there was no lesion; beyond a solitary perforation of the inner coats of the vessel,—if in true aneurism there were no morbid deposits, and the disease was nothing but a simple dilatation,—if there existed no such condition as the atheromatous diathesis, if the facility of coagulation of blood was inversely as its quantity of fibrine,—and lastly, if the anæmic state produced tranquillity of circulation, then, indeed, might we expect good from a mode of treatment which, not content with removing the blood already existing, retards the formation of new blood, and thus depletes the system at both ends, Fortunately, however, for many of the subjects of this treatment, it is often but imperfectly carried out.[1]

[1] In 1830, when discussing the treatment of large internal aneurisms, Dr. Graves and I remarked upon the fact, that a plan of treatment the opposite to that of Valsalva had given great relief in two cases of abdominal aneurism, as recorded by Dr. Proudfoot and Dr. Beatty; and we suggested that the extension of the disease would be better prevented by the use of a generous diet so as to form a more coagulable blood. Since then we have had abundant proofs of the utility of supplying aneurismal patients not only with nutritious food, but even with diffusible stimuli. (Dublin Hospital Reports, vol. v.; see also Dr. Beatty's paper in the same volume.) Dr. Proudfoot's cases will be found in the Edinburgh Medical and Surgical Journal, No. xxii.; and a paper by Mr. King, in Guy's Hospital Reports, should be consulted; see also the Journal Hebdomadaire, No. lxxxii.

It cannot be too strongly insisted on, that aneurism of the aorta, and more especially of the thoracic aorta, at any portion of its arch, is the result of a disease which often affects a large portion of the vessel. We do not deny that in certain cases a temporary relief is produced by the use of the lancet; but this will not justify the practice of repeated bleedings in the ill-grounded hope of effecting a cure. Bertin, indeed, in his "Maladies du Cœur," has well observed that although Morgagni himself, following Valsalva, and again Lancisi, Guattani, Sabatier, Pelletan, Corvisart, Hodgson, and Laennec, have recommended this method; yet that even if we admit their statements, so far as external aneurisms are concerned, there is no proof of the success of the treatment in well-marked aneurism of the aorta. The cases of this disease in which a cure was held to have occurred by these authors are believed by Bertin to have been examples of aortitis or of simulated aneurisms. I have already noticed a case in which, had this method been adopted, the conclusion would have been in favour of the practice: it was probably gouty aortitis.

When it is considered that aneurism of the aorta commonly occurs in persons who have passed the prime of life, or at least the period of youth; that it is a sign of chronic arterial disease, affecting portions of the vessel more or less extensive; that although it does not necessarily produce disease of the heart, it is often complicated with some form of that affection: that pulmonary tubercle is often present, and that anything which tends to weaken the system diminishes the vital resistance to the pressure of the tumour, which pressure again is, from the nature of the disease, augmented by the very causes which weaken the surrounding parts, we have sufficient grounds for condemning the practice even from *a priori* considerations. And, finally, it may be laid down, that there is an utter deficiency of evidence to show that a single cure has ever been effected by the practice in question.

If venesection be employed in the treatment of a thoracic aneurism, it must be, I conceive, only as a palliative measure. Cases

Among the writers on Surgery in this country we may speak of the late Dr. Kirby as the first who pointed out the danger of the antiphlogistic treatment of external aneurism. In his "Cases and Observations," published in 1819, Dr. Kirby has the following remarks:—"The employment of the single ligature, and the medical management of these cases, constitute their most striking peculiarities. In the first three the lancet was not used, nor was any other part of the usual preparatory plan of treatment adopted. After the operations, evacuants were sparingly prescribed, and a regimen more generous than is generally permitted was observed. This practice was suggested by the following reflections:—I had remarked that but few persons recovered after the operation for constitutional aneurism. I thought it not unlikely that these failures might be justly attributed, in some degree, to an error in either the preparatory or subsequent medical treatment, and that something of the want of success was also to be attributed to the disposition of practitioners to experimentalize on human arteries. I did not recollect a single instance in which the patient was not reduced, before the operation, by copious and indiscriminately extensive venesection. The principle which appeared to regulate practitioners in the adoption of these measures I could not explain, except by supposing they were designed to prevent inflammation or to diminish the force of the circulation of the blood. I saw that many who were brought by this treatment to a state of unusual irritability, sunk irrecoverably after this operation,—that others died from mortification of the limb, and some from suppurations. I was led subsequently to the resolution to deviate from the plan I had hitherto seen systematically pursued, and to adapt the treatment to the necessities of each particular case, without reference to any general rule.

"I may be permitted to observe, that practitioners, according to my conceptions, mistook the indications which the pulse afforded. Its fulness, its almost uniform compressibility and frequency, appeared to me to denote a state of the system very opposite to the tonic or inflammatory tendency which has been too often, I fear, supposed to exist."

In Sir Astley Cooper's Lectures we again find these principles of treatment inculcated:—"A gentleman came to town, and was operated on for popliteal aneurism. He recovered in the usual time, no untoward circumstance occurring during his recovery. In twelve months afterwards he became afflicted with aneurism of the aorta, just at its curvature. He came to London, and having been under my care before, he applied to me again. On examination I discovered the aneurism. A consultation was held on this patient in July, when it was agreed that he should be kept low, be occasionally bled, and be always allowed small quantities only of animal food, as it was hoped that by this means the action of the heart and arteries would be considerably lessened. Well, this regimen was prescribed in July; the patient adhered very rigidly to it; but the February following he died from the bursting of an artery into the chest, having lived a shorter period than usual in those cases. Now, it is probable that he would have survived longer had he been otherwise treated; and I will explain to you how it will, no doubt, be met with in which the patient experiences relief of pain, of dyspnœa, or of some other symptom, after venesection; and, in such instances, the practice of occasional small bleedings may be pursued, provided that the general health is not thereby injured. But we are not to conclude from the results in such a case, that venesection should be employed as a general mode of treatment. In determining on the adoption, in the first instance, or the repetition of this method, careful attention is to be paid to the state of the constitution, the force of the heart, and the presence of any irritative or inflammatory action. And there is a greater probability that the treatment will be found useful when the disease is recent, and the impulse not only of the tumour, but of the heart, vigorous.

Local bleeding, on the other hand, is commonly productive of the happiest effects; and it is singular what an amount of relief follows the application of but a few leeches over the seat of disease. These may be repeated from time to time, and in this way we can not only prolong life, but render it comfortable. The use of three or four leeches, whenever the local distress becomes troublesome, is often followed by a period of singular relief; and there can be no doubt that in this way we may delay or arrest the destructive action of a false aneurism. To explain the mode of action here would be difficult; but the success in delaying disease, and from time to time relieving the patient from alarming symptoms, seems to show that local inflammatory action of some kind is, as Hasse teaches, in many cases connected with the extension of the tumour. It may be further a cause of increased vehemence in its pulsations. In several cases we have pursued this treatment for months, and in a few even for years, and thus enabled aneurismal patients not only to enjoy life, but to follow their ordinary avocations in the intervals between each access of distress, whether it was from the stridor, the pain and cough, or even the neuralgic pains which shoot along the neck and affect the back and sides of the head.

Other measures may be resorted to for the relief of pain, such

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is that keeping the patients so low does not agree with them; by keeping them so low, the constitution is rendered irritable, and then whatever is lost in the momentum of the circulating fluid is gained in velocity. I have seen loss of blood in the treatment of aneurisms occasionally useful."

as the application of ice, the external use of chloroform, or of the usual narcotic lotions. The singular effect of issues over the spine, in the case already alluded to, should encourage us to their employment when the erosion of the bodies of the vertebræ is accompanied with tenderness on pressure, or other sufferings. It is unsafe to deduce a principle of treatment from an isolated case; yet in the one now specified it was found, during a long period, that the patient was free, or nearly free, from pain, cough, and dysphagia, so long as the issues discharged freely. On two occasions, the drying of the issues was followed by great suffering, which was again removed on their being re-established. The observations in this case were made with great care.

As a general rule, the diet of an aneurismal patient should be nutritious and even generous. This will apply to cases of aneurism in any portion of the aorta; and the necessity for avoiding a low system of diet will increase in proportion to the duration of the disease and the weakness of the heart. We meet, doubtless, with cases which will, for a time at least, do better upon a low and unstimulating system, but these are exceptional; and we cannot doubt that where the action of the heart is not disturbed by a nutritious diet, it is proper that the patient should have it, and even be allowed a certain quantity of wine or other stimulants. It has often happened to me to be able to remove, again and again in the same patient, the symptoms of stridor, dysphagia, and cough, simply by placing him on a nutritious and even stimulating diet. Most of these cases were in hospital patients, who, when they left our wards, were exposed to want.

A remarkable circumstance occurred in the case already alluded to, of large aneurism with a weak heart, which illustrates the effect of a generous regimen in aneurismal disease. The patient, after a six weeks' course of very low living, became almost desperate from the intensity of the pains, the loss of sleep, and the violence of the throbbings. He was passing through the city when the thought entered his mind that he would have one good dinner before he died. He entered a tavern, and ordered a sumptuous repast of turtle-soup, fish, roast meat, and wild-fowl. Of all these he partook ravenously. He drank a bottle of Madeira, and two glasses of brandy punch; and he declared to me that when he rose from table, he felt perfectly relieved from all his sufferings. He slept well that night, and continued so free from distress of any kind for many days that he thought himself cured of his terrible disease.

Although it has often happened that the stridor from aneurismal pressure has been mistaken for that of chronic disease of the larynx, and further, that tracheotomy has been performed in ignorance of the nature of the disease, yet we should be slow in declaring that the operation of opening the windpipe is unjustifiable in certain cases of aneurism. There is, doubtless, such a case as laryngeal stridor, even though the trachea be compressed by an aneurism; and, under such circumstances, tracheotomy has been known to give a temporary relief. I cannot give the results of any experience of my own on this point; but it appears probable that where the stridor is chiefly from above, or of the mixed kind, and again where it is attended with loss or alteration of voice, the operation might be justifiable as a means of affording present relief.

There is another case in which I have sometimes thought that surgical interference might relieve the patient. I allude to that in which, although the tumour presses strongly against the clavicle, the attachments of that bone to the sternum do not give way. Reasoning from the fact that in some such cases the loosening and semi-luxation of the clavicle is so often followed by a period of extraordinary relief, we might hope that when this yielding did not take place, the division of the attachments of the clavicle to the sternum, if it could be effected with safety, would be followed by alleviation of the internal pressure.

RECAPITULATION.

1. That there are no peculiar or constant signs belonging to aneurism, by which the sounds produced in the tumour can be distinguished from those of the heart.

2. That, as a general rule, the sounds of an aneurism are more and more similar to those of the heart according as the seat of disease approaches the origin of the aorta.

3. That the statement of Laennec, as to the singleness of the aneurismal sound, and that of Hope, who maintains the existence of unequivocal criteria, distinguishing the aneurismal from the cardiac sounds, cannot be admitted.

4. That murmur is frequently absent in aneurisms of the arch of the aorta and the innominata.

5. That the discovery of two centres of pulsation within the thorax, as indicated by impulse and by a single or by double sounds, is the simplest expression of the physical diagnosis.

6. That an extremely weak, almost imperceptible, impulse may attend even a large aneurism of the aorta.

7. That the disease cannot always be detected by percussion.

8. That the double sounds are met with in the true as well as the false aneurisms of the aorta.

9. That the first aneurismal sound is not necessarily, as Hope teaches, a murmur.

10. That we cannot yet explain the presence or absence of murmur in many cases of aneurism.

11. That in certain cases the diagnosis of a communicating aneurism may be ventured on, especially where the signs as indicated by Mr. Thurnam have occurred within a short space of time; and with still more certainty if they have supervened upon some violent effort.

12. That the signs from compression of surrounding parts may be arranged according to their importance, as follows:

a. Pressure on the trachea and bronchial tubes.

b. Compression and sometimes obliteration of blood-vessels.

c. Pressure on the œsophagus.

d. Pressure on the nerves.

13. That the amount of pressure varies within short periods of time.

14. That in consequence of a change in the direction of pressure, parts whose functions had been interrupted or injured may be perfectly relieved.

15. That these circumstances may assist in the diagnosis between cancerous and aneurismal tumours.

16. That in certain cases of aneurismal pressure, the effect on the parts engaged has probably a double origin; on the one hand resulting from mechanical, and on the other from vital causes.

17. That aneurismal may often be distinguished from laryngeal stridor by observing the source and direction of the sound.

18. That a small aneurism, causing lateral compression of the

trachea, will sooner produce stridor than a larger tumour, the pressure of which is more directly from before backwards.

19. That a considerable narrowing of the tube has been produced, yet without stridor.

20. That a degree of the stridor from below may exist, even though the trachea be not compressed, if the tumour engages one of the primary divisions of the tube.

21. That in many cases inequality of vesicular murmur, unattended by signs or symptoms of pulmonary disease, is produced by the compression of one of the bronchial tubes.

22. That this diminution of the vesicular murmur is generally equable over the affected side, but may be confined to the upper portion of the lung.

23. That in certain cases absence of murmur during the first half of the respiratory effort may be observed.

24. That in addition we may enumerate, among the consequences of this pressure on one bronchial tube, the unequal expansion of the sides during respiration; the want of vocal vibration to the hand; and lastly, as Dr. Mayne has observed, the actual contraction of one side, similar to that produced after the absorption of an empyema.

25. That in these cases we may have stridor combined with alteration or loss of voice; lesion of voice without stridor; and, lastly, stridor without any change of voice.

26. That aphonia, strictly speaking, is rarely observed in aneurism; the voice may be variously altered [metaphonia,] but is seldom wholly extinct.

27. That variation of the voice, within short periods of time, is often characteristic of aneurismal pressure.

28. That in the course of the disease, the signs of pressure on the arteries may disappear, and the pulse at the distal side of the tumour be fully restored.

29. That in certain cases permanent obliteration of arterial canals is produced, and in this way important organs, such as the brain and lungs, may suffer from the want of arterial supply.

30. That aneurismal dysphagia may occur without stridor or signs of compression of the arteries or veins.

31. That it may exist to a great degree, and yet the passage of a probang be effected without difficulty.

32. That it may be referred to different portions of the tube within short spaces of time.

33. That in a case where there was evidence of the retreat of the aneurismal tumour from the front of the thorax, and in which great destruction of several vertebræ had taken place, the signs of compression were observed to re-appear on the removal of mechanical support to the shoulders.

34. That the force of the aneurismal diastole cannot be taken as a measure of that of the heart.

35. That in certain cases in which two centres of pulsation are discoverable, the feebleness of the cardiac, as compared with the aneurismal impulse, greatly facilitates the diagnosis.

36. That the existence even of a large aneurism of the thoracic aorta seems to have little if any influence in producing hypertrophy of the heart.

37. That atrophy of the heart may co-exist with a large aneurism; and that the frequent absence of cardiac disease in aneurism facilitates the diagnosis of the latter disease, more especially where the pulsations of the aneurism are attended with murmur.

38. That of the general morbid conditions which accompany aneurism, tubercular phthisis is the most common.

39. That perforation of hollow organs may take place without the patient dying of hemorrhage.

40. That even in cases of external opening there may be many successive hemorrhages, or the blood may ooze out, or be discharged in a small continuous stream.

41. That when the aneurism opens into a free serous cavity, the first gush of blood is not necessarily fatal, nor even the second.

42. That where it opens into a serous cavity in which partial adhesions exist, death may be gradual.

43. That sudden death may occur in aneurism without any rupture of the sac.

44. That when the communication is formed between the sac and the vena cava, or the right auricle, the symptoms of venous congestion are produced.

APPENDIX TO THE PRECEDING CHAPTER.

The following case, by Dr. Mayne, of communicating aneurism, forms an important addition to our knowledge of this lesion. Independent of its value in a pathological point of view, it possesses the great interest of being, so far as I know, the first instance in which the diagnosis not only of a varicose aneurism, but also of the portion of the venous system which had been perforated, was correctly made. The case was one of true aneurism of the arch of the aorta, the entire of which formed a vast pouch which filled up the mediastinum, projecting to the right of the sternum, and encroaching upon the root of the right lung. This disease, in Dr. Mayne's opinion, had been of many years' growth, the dilatation of the vessel occurring so gradually that the neighbouring organs had accommodated themselves to its encroachment. The circumference of this tumour was not less than eleven inches, and its length nine. The patient, a woman aged 50, had been employed in a stooping position when she suddenly felt as if strangled. Her face became immediately livid, her breathing embarrassed, and she felt an indescribable sense of suffocation, accompanied by extreme giddiness. When seen by Dr. Mayne, the following remarkable circumstances were observed. The whole upper part of the trunk was of a deep plum colour, while the lower portion and inferior extremities were pale and almost bloodless in appearance; the eyes were protruded from the orbits, and the face, neck, and upper portion of the chest, were swollen and puffy. All the veins of the head, neck, and upper extremities, were enormously distended, the superficial jugulars being as large as the index finger.

"In a word," says Dr. Mayne, "*all the tributaries of the superior vena cava* were intensely congested, and all the soft parts from which those tributaries spring were swollen and discoloured, whilst *the tributaries of the inferior vena cava, and the corresponding soft parts*, were perfectly free from the slightest trace of congestion, tumefaction, or discoloration."

The principal physical signs observed were a remarkable heaving impulse, single and systolic, to the right of the sternum, commencing at the extremity of the second rib. This was attended with a distinct fremitus, and a loud whirring murmur, which latter was audible all over the chest, its maximum point of intensity being at the sternal end of the second rib, where it conveyed the sensation of being extremely superficial. The impulse of the heart was much feebler than that communicated at the upper portion of the chest; but from the loudness of the purring sound it was difficult to determine whether murmur really existed in the heart itself. On dissection, it was found that the left vena innominata and the superior cava adhered to the surface of the pouch, and the first of these vessels was so completely identified with the diseased aorta that all attempts to separate them were fruitless. The right vena innominata, and the descending cava, were similarly united to the right side of the aortic pouch, and here a free communication had been established between the aorta and the vena cava. Viewed from the aortic side, the orifice resembled the buttonhole of a shirt; it was oval in shape, with sharp, irregular edges, and with a delicate frænum crossing it at about the centre. The heart was absolutely healthy, and all its chambers, valves, and structure, were perfectly normal. "The diagnosis," observes Dr. Mayne, "which I ventured to make in this case was an aneurism of the aorta communicating with the superior cava."[1] It was based upon the following considerations:—

"1st. The congested condition of the entire tree (roots and trunk) of the superior vena cava, and the perfectly healthy condition of the corresponding tree of the inferior cava, showed that the organic source of the mischief was situated somewhere in the course of the trunk of the superior vena cava.

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[1] See Dr. Mayne's communication on Spontaneous Varicose Aneurism, Dublin Quarterly Journal of Medical Science, November, 1853, a paper eminently worthy of study as affording an example of the accuracy of observation and logical deduction which distinguish the various memoirs with which Dr. Mayne has enriched our science. The pulse was similar to that described by Dr. Corrigan in permanent patency of the aortic valves, and a question might arise as to whether it was owing to the varicose communication itself (the competency of which to produce it I do not at all deny,) or was caused by regurgitation into the vast sac during its diastole. To this subject I have already alluded (see page 566.) If experience proves the correctness of Dr. Mayne's observations as well as mine, we shall then have three causes of the regurgitant or collapsing pulse, its most common source being the condition of permanent patency of the aortic valves; next, the existence of dilatation of the thoracic aorta; and thirdly, the occurrence of a communication between the arterial and venous systems within the chest.

"2d. The extensive dulness on percussion in the upper part of the sternal region rendered it almost certain that there was a tumour of considerable size in the mediastinum.

"3d. The extraordinary heaving impulse communicated to the stethoscope, and even to the hand, over the whole of the dull region, and the strength of this impulse, which was vastly greater than that afforded by the apex of the heart itself, appeared to show that this mediastinal tumour was aneurismal.

"4th. The dulness over the superior costal cartilages and intercostal spaces of the right side of the chest in front, with the defective respiratory murmur in the upper part of the right lung anteriorly, rendered it probable that this aneurism had encroached upon the lung; the total absence of any symptoms of pulmonary irritation making against the supposition that there was any special disease either of the lung itself or of the pleura.

"5th. The perfect freedom of the circulation in the carotids and the subclavians made it likely that the aorta itself, and not one of its three great branches, was the source of this aneurism.

"6th. The peculiar whirring *bruit* at once suggested the idea of an abnormal communication between a vein and an artery, for it was a whirr (there is no more expressive term) such as I had never heard before, except in cases of the ordinary traumatic varicose aneurism, or the traumatic aneurismal varix. The site at which this *bruit* was most intensely audible, viz., at the costal cartilage of the second rib upon the right side, and its seemingly superficial source within the chest, forbade the notion of its originating in any valvular disease of the heart; and it certainly sounded very unlike any ordinary aneurismal *bruit*, or even any anæmic *bruit*.

"7th. The suddenness with which the distressing symptoms had set in, but above all, the purring tremor observed to the right of the sternum anteriorly, and also *along the course of the great venous trunks in the neck*, afforded additional evidence, all but conclusive, of the nature of the disease.

"8th. Even the pulse was characteristic, for its 'peculiar jerking feel' depended upon the imperfect distention of the arteries, which resulted from the passage of a portion of the aortic blood into the vena cava through the abnormal orifice. A precisely similar condition of the arteries, although arising from a totally different cause, produces the very same sort of pulse in permanent patency of the aortic valves, the disease so admirably described by Dr. Corrigan."

Sources of Thoracic Pulsation.—Excluding aneurism, the number of cases in which a pulsation distinct from that of the heart exists is not great,—they may be enumerated as follows:—

1. The ordinary pulsation of the aorta in cases of permanent patency. This condition has often been mistaken for aneurism.
2. Throbbing of the aorta, probably from aortitis.
3. Throbbing of the lung, as was observed by Dr. Graves in a case of extensive pneumonia.
4. The existence of a cancerous tumour which, as we suppose, receives a pulsation from the heart or great vessels.
5. The occurrence of an "empyema of necessity," in which a violent pulsation may sometimes be observed.
6. A pulsation communicated to the entire of a large empyematous sac, and derived from the action of a displaced heart in a case of dexiocardia.

On the first three cases we need not dwell: it is important, however, to bear in mind that many of the physical signs of aneurism may occur, although in cases of a totally different nature, such as cancer and empyema.

As the two most common forms of intrathoracic tumour are those of aneurism and of cancer, and as all the symptoms of eccentric pressure are more or less common to the two diseases, the rule is easily derivable that an intrathoracic tumour having been discovered, the diagnosis will be between aneurism and cancer. As the first of these affections, however, is so much more frequent than the second, the chances will be greatly that in any given case of intrathoracic tumour the disease is aneurismal.

But I have already shown that a cancerous tumour may not only produce all the symptoms which we observe in aneurism, as proceeding from compression of surrounding organs, but that it may exhibit a diastolic pulsation, accompanied by a distinct bellows murmur. As might be expected from the rarity of the disease, our experience of those cancers which simulate aneurism is as yet but limited. I am aware, however, of two cases of cancerous tumour: the one presenting only the signs of surrounding pressure, without pulsation or murmur; the second having the latter signs, in addition to those of pressure on surrounding parts. This case has been already commented on.[1]

Of the first form the following case is illustrative:—Some years ago a man was admitted into one of my wards to be treated for chronic laryngitis. He was emaciated and cachectic; but it was easy to observe that the stridulous breathing was similar to that so often seen in aneurism. It was the stridor from below. The upper portion of the sternum and the clavicles were dull on percussion. I came to the conclusion that a tumour of some kind existed; further consideration made me incline to the opinion that the disease was cancerous: for, had the stridor proceeded from an aneurism sufficiently close to the surface to cause dulness of the upper portion of the chest, we should have had some of the acoustic or tactile signs of that disease; but no pulsation, double sound, nor murmur, could be discovered. This patient had two kinds of expectoration, perfectly distinct from one another: one a muco-purulent fluid, similar to that of chronic bronchitis or phthisis; the other, a creamy liquid of a milk-white colour, which, though sometimes combined with the ordinary expectoration, was often secreted in great quantities without any admixture. On dissection, numerous cysts, varying from the size of a hazel-nut to that of a pullet's egg, were found surrounding and compressing the trachea just above its bifurcation, and also embracing the œsophagus. These cysts contained a milk-white fluid exactly similar to that expectorated. The upper portions of both lungs contained encysted matter of the same character, but no direct communication between the cysts and the bronchial tubes could be discovered. In the larger ramifications the milky fluid was found in abundance; but in the more minute tubes its consistence approached to that of coagulable lymph.[2]

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[1] See my researches on the Diagnosis of Cancer of the Lung, Dublin Journal of Medical Science, vol. xxi.

[2] Although no microscopic examination was made of the white fluid expectorated, and also found in the bronchial tubes, its similarity to that contained in the numerous cysts was complete. I hardly entertain a doubt that it was a cancerous secretion; and that in this case that law of vicarious action was illustrated, by which, as in instances of empyema or of hepatic abscess without direct communication, the bronchial tubes secrete a fluid in all respects similar to that in the pleura, on the one hand, or the abscess of the liver, on the other.

In a report of a case of pulsating cancer, published in 1842, I have mentioned as a diagnostic in favour of cancer, the discrepancy between the extent of dulness and the force of pulsation. A similar observation has since been made by Dr. O'Ferrall.[1] In his case there was a double murmur at the base of the heart, a diminished pulse in the right radial artery, dysphagia, and ultimately stridor. He leaned to the opinion that the disease was cancerous, and not aneurismal. In a case by Dr. Law the tumour compressed, and obliterated the right vena innominata, and greatly interfered with the superior cava. The tumour was traversed by the arteria innominata.[2]

From what we now know of the disease, I believe we could recognise (under a similar combination of circumstances) a pulsating cancerous tumour. The following points are of importance in settling the question between the two diseases:—

1. *The Extended Dulness.*—This would show that the tumour, whatever was its nature, was not only a large one, but that it must be near the surface. Now, if under these circumstances, the diastolic throb has much less force than in aneurism, a probability would be created that the disease is a pulsating cancer.

2. In a great number of aneurisms of the arch of the aorta the pulsation is distinctly double. I apprehend that in the cancerous disease the second or back stroke either does not exist, or is extremely feeble.

3. If we suppose a case in which the evidences on both sides were nearly balanced, the existence of a soft, single, and systolic bellows murmur should incline our opinion towards cancer; bellows murmur in aneurisms of the arch is a more rare circumstance than has been supposed.

We are still in want of observations which would fully explain the cause of pulsation and murmur in these tumours. I am at present of opinion that the throb is communicated by some vessel or vessels, which either impinge on the tumour or run through the mass, and that, owing to its semi-fluid consistence, a species of diastolic throb is thus given. It is true, that in some cases of malignant osteo-sarcoma the tumour has an expanding pulsation, similar to what might be anticipated in an exaggerated aneurismal varix or pulsating bronchocele; but nothing of this kind has been observed in thoracic, or, as far as I have seen, in abdominal cancers. In fact, these structures possess so few and such delicate proper vessels, that this kind of pulsation could not be expected to occur.

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[1] See Transactions of the Pathological Society of Dublin, April, 1842.

[2] See Transactions of the Pathological Society of Dublin, January, 1844.

We cannot, on the other hand, lay it down that in all cases of cancerous tumours engaging the great vessels there is simulation of these symptoms of aneurism. There may be only those of pressure, as occurred in a case of tumour in the anterior mediastinum observed by Dr. Adams. The tumour was, however, of a very hard consistence, though containing here and there masses of cephaloma.

In contrasting the phenomena of pressure in the two classes of intrathoracic tumours—namely, the aneurismal and the cancerous—we observe certain differences. In cases of aneurism there is nothing more remarkable than the evidences of a varying pressure. This variation is twofold:—1st, as having reference to a varying amount of pressure on the same part at different times; and 2nd, as showing a complete and permanent change in the direction of the pressure.

It has been shown that the stridor from below, and dysphagia from pressure, may appear and disappear either wholly or in part during the progress of a case of aneurism, or the direction of pressure may altogether change; so that a tumour which is evident on the front of the chest may wholly disappear or present posteriorly. Now we find that this character of variation, either in the amount or direction of pressure has not been observed in cancerous tumours, which are either stationary or exhibit a slowly progressive advance.

We are not to understand from this that variation is the constant character of aneurism, but merely that its marked occurrence should lead to the diagnosis of aneurismal rather than of cancerous tumour.

Another point of difference is the frequent occurrence of varicose veins in cancer, and their comparative rarity in aneurismal disease. Whether in these cases the veins are themselves obstructed by cancerous matter is still to be determined, but we may perhaps explain the circumstance by referring to the generally healthy state of the lung in aneurismal and its diseased condition in cancerous tumours. The venous trunks are in the one case compressed against the soft and elastic lung, and the force of the compressing agent itself is continually varying; whereas, in the pressure from a cancerous tumour, the lung being often invaded by the disease, the vein is exposed to a more constant pressure against a more resisting medium, and the collateral circulation, thus rendered necessary, may be observed even at an early period of the case.

Pulsating Empyema.—In cases of this kind it is unlikely that any difficulty would attend the diagnosis. Two instances, in which the sac had become bilocular, so that the external tumour formed the pulsating mass, occurred to Dr. Graves, and have been reported by Dr. Macdonnel.[1] I have lately observed the case of pulsation of the entire sac by a vast empyema, in which the heart was displaced far to the right side. In this case paracentesis was performed on three occasions, and large quantities of fluid, on each operation appearing more and more purulent, were withdrawn. Before each tapping, every stroke of the displaced heart produced the most extraordinary diastolic pulsation of the whole of the left side. The bed was shaken at each beat of the heart, and the patient's sleep interrupted by the vast and violent throbbing of the side; yet the force of the heart did not seem much augmented. There was no appearance of any external pointing of the contents of the sac; and it was remarkable that notwithstanding the evacuation of the fluid on three occasions, the heart never moved from its situation at the right of the sternum.

Diagnosis between true and false Aneurisms, and between Aneurisms of the Innominata and of the Arch of the Aorta.—With respect to the first of these questions, I have already suggested that the long-continued existence of the physical signs of a pulsating tumour, without the occurrence of the usual suffering which attends false aneurism, might lead us to suspect that the case was one of simple dilatation of the artery. This subject has been since handled by M. Gendrin and Dr. Lyons, the latter of whom has expressed his belief that no additional diagnostics have been as yet discovered.

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[1] See Dublin Journal of Medical Science, vol. xxv. p. 1.

We owe to Dr. Holland an important memoir on the diagnosis of aneurisms of the innominata.[1] From an examination of many recorded cases, he concludes that the disease may be distinguished from aneurisms of the arch, principally by the circumstance that almost all its symptoms and signs have a tendency to occur at the right side of the body, while those of aneurisms of the transverse portions of the arch of the aorta are met with, at least in the first instance, at the left side.

Existence of Aneurism without manifest Physical Signs.—To those whose knowledge of aneurism is derived from written descriptions rather than bed-side experience, it may appear strange that a disease of such importance should ever exist without being accompanied by signs, which if not sufficient to determine the nature of the affection, would, at all events, indicate some important organic disease. Yet cases do certainly occur in which, at some period at least, the disease may elude the most careful investigation; and proof is not wanting of the continuance of this latency even after a copious hemorrhage has taken place from the sac itself. It is probable that some of these instances, in which all physical signs have been wanting, were examples of true rather than of false aneurism. But this is merely a suggestion.

From what I have seen, I would divide the cases which were deficient in physical signs into three classes. In the first there was a true aneurism of the arch of the aorta, elongated and fusiform, with or without local dilatations. A case of this kind has been given in which there was no symptom beyond the doubtful one of the tussis clangosa. The disease was complicated with extensive tuberculization of the lung.

Of the next case we have also given an example. Here, after the existence of manifest physical signs and symptoms of aneurism presenting in the front of the thorax, the extraordinary circumstance occurred of the disappearance of all physical signs of aneurism; so that, had this patient been examined at that juncture by an observer ignorant of the previous circumstances of the case, and who trusted in negative results as proving the absence of aneurism, the patient would have been declared free from the disease, although at the time there existed a vast false aneurism which had, as it were, formed a chamber for itself at the expense of the bodies of the vertebræ.

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[1] See Dublin Quarterly Journal of Medical Science, vol. xii.

The last case may be that where in a man of large stature and deep thorax a small aneurism has formed, of such a volume, and presenting in such a direction, that it does not interfere with the trachea, bronchial tubes, or œsophagus. In a case of this kind it is possible that the occurrence of deep-seated thoracic pains, or, as in an instance with which I am acquainted, of a sudden and copious hæmoptysis, neither preceded nor followed by symptoms of pulmonary disease, may lead to the suspicion of an aneurism; and the practitioner must be cautious not to allow the absence of all physical signs of the malady to make him declare that the patient is free from arterial disease. The error of declaring the absence of organic disease, in consequence of the want of physical signs, has led many of our brethren into disagreeable positions; and it must not be forgotten that in physical examinations negative results may furnish significant indications; indeed nothing should more awake our attention than the occurrence of important symptoms without there being signs to account for them. In the early stages of phthisis, the want of commensurate physical signs gives a fearful importance to the symptoms of cough, hæmoptysis, pain, and irritative fever; in gangrene of the lung, the same deficiency of physical evidence indicates a case in which the fetid breath and putrid expectoration will probably be repeated, until at last an eschar is evident; and in suspected aneurism, the absence of all signs of tumour, especially when certain symptoms have been produced, will not justify our declaring the patient in an absolutely safe position.

CHAPTER XII.

ANEURISM OF THE ABDOMINAL AORTA.

Our knowledge of the diagnosis of this affection may safely be said to date from the year 1830, when Dr. Beatty, of this city, published his accurate observations on a single case of the disease. Although Morgagni[1] had shown that the lesion had been often mistaken or overlooked, while Laennec had indicated the physical signs of aneurism, yet it was not until after the publication of Dr. Beatty's record of a case of aneurism of the abdominal aorta[2] that physicians were enabled to diagnose that disease, or even led to suspect it in cases which before had been differently interpreted. We must not lose sight of the fact, that in the years 1827, 1828, and 1829, as is shown by Dr. Beatty, an individual labouring under this disease, with marked and violent symptoms, was carefully and repeatedly examined by the most eminent physicians in Dublin, London, and Paris, and yet without a suspicion being formed of the real nature of the case. And when we find such names as those of Graves, Cheyne, Brodie, Colles, Townsend, Wilson Philip, and Andral, among those in attendance on the patient, we can come to no other conclusion than that, up to the period in question, the diagnosis of the disease, at least from its symptoms, had never been established.

We may, then, date our knowledge of this part of medicine from the publication of Dr. Beatty's case, which, in addition to its other important characters, is above all valuable, from the fact that the patient was under the inspection of an accurate observer from the earliest stage of the disease, so that no symptom or change escaped notice.

Although, in most cases, the general character of the symptoms is the same, yet, as might be expected, the violence of pain, its remissions or intermissions, and the seat of suffering, vary in different individuals. As the case above alluded to presents the most graphic account of the symptoms, it will be proper to give an abridgment of it here, for it is, in fact, the key to the whole subject.

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[1] "De Sedibus et Causis Morborum." Cap. "De Doloribus Lumborum."

[2] A Case of Aneurism of the Abdominal Aorta, by Thomas Beatty, M. D. Dublin Hospital Reports, vol. v.

A barrister, aged 33 years, of a robust frame and temperate habits, was, in 1825, attacked with dull pain in the loins, which was held to be lumbago. The pain he described as deep-seated, and between the bowels and the spine. The slightest sudden change of position produced acute pain, shooting from the loins towards the spine. The pain gradually extended round the belly, which became tympanitic; and was often relieved by the patient assuming the erect position and discharging wind upwards. The pulse and appetite were good, but pain commonly supervened on eating. During the next three months he had some remarkable intermissions of the suffering, during which he appeared quite restored to health; when, early in November, the pain returned with increased violence, affecting both sides. It now assumed the form of severe spasmodic twisting of the intestinal tract, but particularly in the region of the colon, and produced the greatest torture. He also suffered from a more permanent dull pain, distinct from the spasmodic pain, which seemed to affect the whole abdomen, pulling it from every part. This uneasiness was always worse in bed, and continued to increase the longer he lay. It was increased by external heat. During the winter the disease continued with remissions, and no relief was obtained except from the use of anodyne injections; but the distress afterwards produced by the costiveness which they induced obliged him to discontinue their employment.

"He was not at any time, however," says Dr. Beatty, "incapacitated from attending to business in court. At one period during the term, the effect of speaking in an important cause was very striking: he had been suffering greatly during the whole day, and could with difficulty remain in court; but when he rose to address the bench, all pain fled, and he continued perfectly well while speaking, and during the following evening."

In about three months the pain, which had been latterly felt most in the left side, changed to the right iliac region, and the patient was attacked with evening rigors, which were occasionally severe and protracted. The least accumulation in the bowels brought on attacks of the pain and spasms; and on two occasions the pain affected the left loin, hip, and thigh, with frequent violent spasms shooting forward in a straight direction to the belly. The bowels were much constipated. His general health at this time improved, but he became affected with cramps in the feet and legs at night; and any return of the pain in the side or uneasiness of the bowels, was attended with convulsive twitching of the legs, particularly when the patient lay down. His sleep was constantly broken by colicky pains. At this time he proceeded to Paris. During his stay in that city he improved in every respect. His appetite, spirits, and flesh, returned; he ate and drank without restraint, and his sleep was sound and refreshing.

"He continued well during his journey back to England, almost up to the very moment of his landing at Dover, when suddenly, while walking the deck of the packet with Dr. Townsend, who accompanied him, a most violent pain shot through the back and bowels, the effect of which was to throw the whole of the belly into a state of spasm and torture, and in this state he continued for several hours." This attack was followed by another intermission, and new hopes of recovery were formed, when a fresh access of pain, now, for the first time, extending to the chest, made its appearance. The pain was confined to the lower part of the sternum. Next day it moved to the right side, and occupied exactly the region of the liver; and he complained of severe pain in the right scapular region. Any attempt at the horizontal posture brought on an increase of suffering. The pulse continued steady at 80; but blood drawn from the arm exhibited the inflammatory crust. In a week he was able to leave his room, and the pain on the right side gradually subsided. "He had resumed," says the narrator, "the amusement of singing, on his return to Ireland, in which art he was an admired proficient, and his evenings were now usually enlivened by this recreation. I mention this to show how little the respiratory function was interrupted. In the course of a fortnight he was so far recovered as to be able to use exercise on horseback, which he continued until early in December."

"From this time he had repeated attacks of his disease, only relieved by the use of opium; and in the middle of January the pulse, for the first time, varied from a state indicative of perfect health. It rose to 100, and never fell again. "On the 17th it was thought advisable to salivate him with mercury, and the large doses of opium were suspended; but no language could describe the agony he suffered that day. The spasms of the back and side came on with such violence as frequently to force him to cry out, and, leaping from the chair on which he sat, to throw himself flat on his face on the bed, in which posture he obtained a partial and temporary relief. His screams at this time, though he was naturally a strong-minded man, were heard from top to bottom of his house."

The constitution now rapidly gave way; the paroxysms continued, throwing the muscles of the back and belly into a state of tetanic spasm. He suffered from dysphagia; panting respiration followed the attempt to swallow; and when fluids were taken, a gurgling noise was heard in the chest. The liver appeared to be distinctly enlarged, and increased daily in size; and no relief was obtained except by the use of enormous doses of opium. Towards the close of the case, his daily dose of black-drop was from 150 to 200 drops; and on one occasion it amounted to 285 drops; and notwithstanding the great amount of opium, he was never drowsy or apparently narcotized, though sometimes excited and slightly delirious. He died on the 26th of February, 1829, the disease having ran its course in about eighteen months.

A large aneurism, covered by the crura of the diaphragm, which were expanded and stretched tightly over the surface of the sac, was found lying on the last three dorsal vertebræ, the bodies of which were deeply eroded. A well-defined opening, as large as a shilling, and a little above the origin of the cœliac artery, was the communication between the aorta and the sac, which latter had burst into the left pleura by an irregular rent on its upper portion.

The apparent enlargement of the liver was found to have been owing to its dislocation, for it had resumed its natural situation, and was found very little increased in size, and in structure quite healthy. Its outer convex surface was marked by deep indentations, from the strong pressure against the ribs.[1]

If we review the symptoms in this important case, we shall find that they constitute a group not referrible to any of the ordinary diseases of the solid organs or digestive tube. We should consider carefully:—

1. The occurrence of deep-seated pain in the back, with neuralgic exacerbations following any change of position.
2. The extension of this pain to the intestinal tract, producing dreadful agony, similar to that in painters' colic.
3. Its complication with muscular spasms of the lower extremities.
4. The absence of fever.
5. The occasional complete intermissions of pain.
6. Its obstinate resistance to every variety of general and local treatment.
7. The occurrence of displacement of the liver, simulating its enlargement, at an advanced period of the case.

It is superfluous to observe, that we have here a group of symptoms which can only belong to aneurism. In this case, the true nature of the disease being never suspected, no stethoscopic examination was instituted; but it is by no means certain that the nature of the disease would have been discovered even with the aid of auscultation; for if the aneurism had been one of those in which murmur is absent, a circumstance so common when the disease is situated in the thoracic aorta, its detection by physical signs might have been impossible. The aneurismal tumour was never felt, so that in the then existing state of knowledge, the opinion of Andral, that the case was an example of a rare form of intestinal neurosis, was perfectly reasonable.

GENERAL HISTORY OF THE DISEASE.

Abdominal aneurism appears principally to affect persons between the ages of 25 and 40, and is much more common in the male than the female. Its ordinary form is that of false aneurism, as we might expect from the extraordinary suffering which so commonly attends it. Very little is known of the history of true aneurism of the abdominal aorta.

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[1] I have given this case in so condensed a form that some interesting particulars have been omitted; but I would earnestly counsel all students of arterial disease to read it as originally published. It is, I repeat, the source of all our knowledge of the symptoms of this disease.

Three modes of termination of life may be enumerated in this affection:—

1. Sudden death by rupture of the tumour into the sac of the peritoneum, or of the pleura, into some portion of the digestive tube, or into the lungs themselves.

2. The formation of a diffused aneurism by the opening of the sac into the retro-peritoneal tissue, or into the omentum; the patient sinking gradually from the effects of loss of blood, and slow irritative fever.

3. Death from exhaustion, induced by long-continued suffering, without any rupture of the sac. This I have only observed in one instance, where the aneurismal sac was of extraordinary size.

It may be laid down as a general rule, that the sufferings will be greater in proportion as the disease arises higher up in the tube.

There is no necessary connexion between aneurism of the abdominal aorta and disease of the heart. The combination is a rare one; and I have even seen, in a case of vast abdominal aneurism, in which the sufferings of the patient from pain and the violence of the pulsations were extreme, the heart in a state of atrophy, and diminished capacity, very similar to that in cases of protracted phthisis. Nor is extensive arterial disease by any means as common as in thoracic aneurism. The disease may exist without any discoverable aortic lesion, save the single circumscribed perforation which led to the formation of the sac. The pulse and action of the heart are often perfectly undisturbed and tranquil, at least until the advanced periods of the disease, when the health gives way under long-continued suffering. A species of low irritative fever may be then established, particularly if the aneurism becomes diffused.

And this leads to the first great point in the diagnosis of this terrible disease, namely, the disproportion between the violence of the symptoms and the amount of constitutional disturbance. It is this circumstance which has so long obscured the subject. We commonly find the functions of respiration, circulation, digestion, secretion, and nutrition, well performed, although the disease is not only in actual existence, but, from time to time, inducing paroxysms of dreadful and protracted suffering.

In the next place, the attacks of pain are not accompanied by inflammatory fever; on their subsidence the patient regains his usual health, and no trace of organic change of any of the great viscera, nor even lesion of function, can be discovered. So far the disease has the character of neuralgia; and there is no doubt that the pain is neuralgic, but induced by a condition of parts acting, *ab externo*, on the nervous cords.

It is, therefore, to be laid down as a practical rule, that in all cases of violent neuralgic affections engaging the region of the diaphragm, the dorsal or lumbar spinal regions, or the abdominal viscera, the patient should be examined for aneurism, more especially if the disease be remittent, and the general health in the earlier periods unaffected.

This disease has been mistaken for the following affections:—

1. Rheumatism, affecting the lumbar muscles, diaphragm, and spine.
2. Disease of the kidney, with or without calculus.
3. The presence of worms.
4. Flatulent colic.
5. Intestinal neuralgia, analogous to painters' colic.
6. Psoas abscess.
7. Caries of the spine.
8. Disease of the liver.
9. Malignant tumours of the abdomen.

In two of these cases, namely those in which psoas abscess and hepatic disease are supposed to exist, the erroneous opinion may receive a great degree of strength from the evidences of organic diseases in the very regions where the affection was held to exist. It happens, as has been shown by Sir A. Cooper, Professor Harrison, and myself, that in certain cases where the aneurism becomes diffused, tumours, soft and fluctuating, but evidently deep-seated, arise in the lumbar regions: and though in reality they are vast effusions of blood, they might be easily mistaken for deposits of purulent matter. Nay, the tumours may point at Poupart's ligament, as occurs in psoas abscess, and be attended with lateral and even posterior curvature of the spine.

Again, the advocates for the existence of disease of the liver might find a corroboration of their opinions on discovering at a certain period of the case that an hepatic tumour had become manifest. This occurred in Dr. Beatty's case, in which an apparent enlargement of the liver appeared nearly a year and a half after the commencement of the disease. But the appearance was deceptive, and caused by displacement of the organ from the pressure of the aneurism. On the rupture of the sac, the liver returned to its natural situation. I have recorded a case of aneurism of the hepatic artery, in which, also, this simulated enlargement of the liver was observed.

Let us now examine other examples of the disease, which, though they vary in their symptoms, and in the mode of death, yet partake of the generic character so well marked in the case recorded by Dr. Beatty.

With a view to practical utility, we may divide these cases into certain categories. For example:—

a. Aneurism of the abdominal aorta, combined with symptoms of disease of the heart.

b. Aneurism, causing death by rupture into serous membranes.

c. Aneurism, causing death by opening into the lungs, or some portion of the digestive tube.

d. Aneurism, simulating psoas abscess.

e. Aneurism, with successive openings.

f. Aneurism, without erosion of the vertebræ.

g. Aneurism, simulating cancerous tumour, and bursting into the omentum.

We have seen that the combination of aneurism of the abdominal aorta with disease of the heart is rare. It is much more rare than that of thoracic aneurism with the same lesion. Even in this latter case, the combination of heart disease is by no means constant, and the contrast of the tranquil and natural impulse of the heart, with the violent pulsation which attends on aneurism, is often most striking in both forms of disease. It may be added, that the combination of extensive aortic disease is much more rare in abdominal than in thoracic aneurisms, especially those of the innominata and the arch of the aorta.

CASE LXXIV.—*Aneurism of the Abdominal Aorta; Double pulsation of the Tumour and displacement of the Heart.*[1]

A man, aged 29, after exposure to cold, was attacked with severe lumbar pains, accompanied by a fit resembling convulsive colic. The pain presented exacerbations, and was then pulsative —the pulsations being referred to the left iliac fossa. The patient uniformly experienced relief by lying on his face and right side, while any attempt to turn on the back or left side produced great pain. In about a month he was attacked with pain in the region of the heart, succeeded by palpitation, which continued up to his admission, four months having elapsed from the commencement of the disease.

On admission he was pale and exsanguine, with a distressed countenance; the impulse of the heart was greatly increased, and apparently communicated from an extensive surface. It was augmented by any change of position, but particularly by turning on the back or left side, when it could be felt over the whole front of the chest and epigastrium. Its pulsations were extensively audible, and, during excitement, a bellows murmur accompanied the ventricular sound. Under the influence of complete rest, a very low diet, and local bleeding, the excitement of the heart was completely reduced; but still, whenever he turned on the left side, the increased action re-appeared, subsiding again to its natural standard when he resumed his usual position.

On the eleventh day after his admission, a pulsating tumour made its appearance on the left side, on a level with the last dorsal vertebra. The pulsation occupied a space of about two square inches, which was tender to the touch; it was not accompanied by bellows murmur; but this sound could be heard in the epigastrium. Pressure on the tumour caused nausea, and a sensation of fluid ascending towards the chest.

As the tumour gradually increased, the pain and general distress diminished. He left the hospital in an improved condition, but was re-admitted after six weeks, the pain having greatly increased. He compared it to a sensation as if the bones of the pelvis were separating. The trunk was bent to the left side.

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[1] Clinical Reports of Cases in the Medical Wards of the Meath Hospital, by Dr. Graves and Dr. Stokes. Dublin Hospital Reports, vol. v.

A distinct double pulsation was now observed in the tumour, with two corresponding sounds; the first dull, the second clear. These signs bore the greatest resemblance to those of a hypertrophied and dilated heart.

In a short time we observed that the heart was displaced; at first it was found to beat in the epigastrium, the impulse having left the usual situation. In the course of a few days it became feebler in the epigastrium, but could be felt pulsating on the right side, at the sternal end of the fifth rib, and ultimately became fixed in the intercostal space of the third and fourth ribs. Here the sound of its contractions was plainly audible, while in the epigastric region they could scarcely be heard.

Soon after this occurrence he again left the hospital, and indulged freely in spirituous liquors; violent action of the heart supervened, and on the following day a dreadful fit of convulsions, with pain in the abdomen and back, came on, during which he suddenly ceased to exist.

His death took place in three months from the appearance of the tumour. Dissection was not allowed.

In the original report of this case by Dr. Graves and myself we expressed our opinion, that the chief, if not the only, cause of the double pulsation of the sac in this case was the pressure of the tumour on the dislocated heart. I do not now feel so certain on this point, as the occurrence of double pulsation has been so commonly observed in large aneurisms of the thoracic aorta. I am not aware, however, of any instance, except that before us, in which double pulsation has been observed in abdominal aneurism, a circumstance very difficult of explanation. It cannot, on the other hand, be denied that the impulse of the dislocated heart may also have acted on the doubly pulsating tumour; and I would here refer to the case of pulsating empyema, with dislocated heart, which has been already described, as showing the singular influence of the heart's action on fluid contained in a sac external to, and pressing upon, that organ.

The perfect similarity, however, of the signs in this case to those of thoracic aneurisms in which there is no pressure upon the heart, leads me to think that much, at least, of the double pulsation arose independently of any lateral impulse communicated by the heart. It is probable, also, that the aneurism engaged both the abdominal and thoracic cavities:—a mixed case of which Dr. Law has given an example in a communication to the Pathological Society, and which has since been noticed by Dr. Lyons.

The influence of pressure on the sac, in producing disturbance of the heart's action after its permanent excitement had been reduced, was a singularly interesting, and, to us, novel fact.

Finally, we may remark, that that species of retrograde nervous influence, supposed to be induced by aneurismal disease, upon the heart's action, seems to be rare in abdominal aneurism, in which, as I have before stated, the heart's action is often perfectly undisturbed, at least until the latter periods of the disease.

The death, by convulsions, is worthy of remark. These were obviously of that nature induced by sudden and great losses of blood. I have already detailed a case in which several distinct eruptions of blood took place from an aneurism, each of which was accompanied by a convulsive paroxysm, in the last of which the patient died.

Is it owing to the suddenness of the loss or to the quantity of blood effused, that death is produced in cases of aneurismal rupture? I think that, in many cases, at least, the influence of the former is the more important.

CASE LXXV.—*Aneurism of the Abdominal Aorta; Hemorrhage by successive gushes.*

This example occurred under the observation of Dr. Lees.[1] A man, aged 48, had, for two years, been in bad health. His illness commenced with acute pain in the loins and distressing sensations, attributed to flatulent distention and debility, with frequent vomiting and anorexia. He latterly suffered from cough, palpitation, dyspnœa, *and difficulty of swallowing*, the effort causing severe pain. He was emaciated and had a cachectic appearance. The pulse was 120, small and feeble, while the action of the heart was strong and excited. The abdomen was retracted. The lower portion of the chest sounded dull posteriorly, from the sixth rib downwards, with a feeble respiratory murmur.

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[1] See the Proceedings of the Pathological Society of Dublin.

In the left hypochondriac region, a round, hard, incompressible, and apparently immovable tumour was felt, presenting a well-marked diastolic pulsation, most distinct at its inner and outer portions. Here, when the patient was in the recumbent position, a distinct and sharp bellows murmur existed. It was not propagated along the course of the aorta, nor discernible at the inner portion of the tumour. The pulsation of the tumour, though manifest to the eye and hand when the patient was in the recumbent position, almost disappeared on his sitting up, and the bellows murmur was no longer heard.

The diagnosis in this case presented some difficulty, as the appearance of the patient, the hardness of the tumour, and the absence of lumbar pain, seemed to indicate a cancerous disease. On the other hand, the diastolic pulsation, strongest in the recumbent position, the peculiar character of the murmur, and its total disappearance in the erect position, induced Dr. Lees to diagnosticate aneurism.

For three days previous to his death this patient had each day a fainting fit. He died suddenly after a muscular exertion.

The aneurism lay under the left lobe of the liver, depressing the pancreas. It had burst into the peritoneal cavity, and had eroded the bodies of the eleventh and twelfth dorsal vertebræ.

In this case Dr. Lees is of opinion, that the hemorrhage occurred by successive gushes which corresponded to the fainting fits. And there is much reason to concur with him in this view.

In another case of abdominal aneurism, which occurred in the Meath Hospital, the patient fainted while under examination, and on his recovery Dr. Lyons observed *that the left side had become dull as high up as the lower edge of the scapula.* He at once made the diagnosis of the bursting of an abdominal aneurism into the left pleura. The patient lived twenty-four hours after the rupture of the sac; and the diagnosis was verified by dissection.

We owe to Dr. Law the first observation of the newly-developed physical signs in rupture of an aneurism of the abdominal aorta, and also that of the hemorrhage by successive gushes. A patient of his presented the usual symptoms of the disease. A distinct diastolic pulsation, and bellows murmur were present. On one occasion, after rising from bed, he was attacked with a rigor, which lasted for twenty minutes, after which it was found that the murmur and pulsation had disappeared. The left portion of the abdomen had become tender to pressure, part of it giving a dull sound on percussion and part a tympanitic resonance. In the course of twelve hours he was again examined, and the pulse was found absent. On the following day a diffused bellows murmur was perceptible in the epigastrium. The pulsations of the heart were rapid, and with but a single sound. The femoral pulse was distinct. Next day he was better, but an obvious fulness existed between the spine of the ilium and symphisis pubis. He expired suddenly on this day, the fourth day from the first appearance of hemorrhage.

The aneurism was bilocular, communicating with the aorta at the cœliac axis, where the vessel was dilated. It had become diffused. Two pounds of blood were extravasated behind the peritoneum; and the effusion had made its way into the left pleura.[1]

In this case we observe the somewhat rare occurrence of sudden death by rupture into a serous cavity, supervening on a diffused aneurism, with hemorrhage into cellular structure. The latter was probably pointed out by the rigor, followed by the sinking of the heart's action, and the cessation of the murmur and pulsation, while the rupture of this diffused aneurism into the pleura was followed by sudden death.

We have seen, however, that even when the tumour opens into a free serous cavity, the first or even the second hemorrhages may not be fatal.

The occurrence of pulsation in the femoral arteries, on the third day after the rigor, and coincident with the return of the bellows murmur in the epigastrium, is of importance as bearing on the question,—whether, by the absence or presence of femoral pulsations, we may distinguish between the rupture of an abdominal aneurism and other solutions of continuity, such as peritonitis, from perforation of the digestive tube.

Some years ago a patient was brought into my wards in a dying state. It appeared that for more than a year previously he had laboured under pain, referred to the region of the stomach, which was occasionally severe. On the day before admission, while walking through the streets, the pain became suddenly aggravated, and he fell, and remained in a state approaching to syncope for a considerable period. No re-action seemed to occur. When I saw him the abdomen was swelled, but soft, and the patient bore pressure well over every portion of it. The extremities were cold, the face sunken, and the pulse at the wrist imperceptible.

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[1] Proceedings of the Pathological Society of Dublin, April, 1843.

That some solution of continuity had taken place here was manifest; and the case had many of the appearances of hemorrhage from an abdominal aneurism. The only other view to be taken was, that the intestinal tube had suffered perforation, and that the case was one of those rare examples in which the consequent peritonitis was either from the first unaccompanied with tenderness, or that the absence of pain on pressure was caused by the sinking condition of the patient.

I now examined the state of the femoral arteries, and found the pulsations at the groin not only distinct but excited; it was, therefore, obvious that the failure of the pulse at the wrist could not be from hemorrhage; for this cause would act equally on all parts of the arterial system. I, therefore, diagnosed perforation of the intestine, attributing the increased action of the femoral arteries to the excitement of peritonitis. The diagnosis proved in the main correct; but the perforation was from an ulcer in the stomach.

CASE LXXVI.—*Aneurism of the Abdominal Aorta; Death by Perforation of the Lung and Hæmoptysis.*

A middle-aged man had for eighteen months suffered from pains referred to the dorsal and lumbar regions. He was seen by Mr. Pakenham, who first detected the nature of the disease, and subsequently by me in consultation with that gentleman. He complained of intolerable pains referred to the last three of the dorsal and first three lumbar vertebræ. This pain was aggravated by motion, and relieved by the patient lying with his back turned to a strong fire, or by lying on the face. It was increased by pressure. The two forms of pain, as noticed by Dr. Law, existed in this case,—one, fixed and dull; the other, lancinating and paroxysmal, often shooting downwards into the scrotum; and it appeared that this latter was not perceived until three months after the invasion of the fixed pain.

A deep-seated pulsating tumour existed under the cartilages of the left lower ribs. Its impulse was synchronous with the diastole, or, in other words, with the second sound of the heart. A distinct interval elapsed between the systole of the heart and the throb of the tumour. When deep pressure was made to the left of the epigastrium the pulsation of the sac appeared to be violent. It was attended by a bellows murmur, which, however, was inaudible posteriorly; pulse 80, and regular. On the day before his death his countenance was haggard, the pulsations of the tumour had greatly increased in force, and the pulse was rapid. After expectorating a great quantity of blood, the patient died in a state between asphyxia and syncope.

The left lung was found engorged to the last degree with blood, which also filled the bronchial tubes. A laceration of the lower lobe of the lung, where it had been in contact with the sac, was discovered, corresponding to the opening of the sac. The aneurism, seated under the crura of the diaphragm, had passed upwards between them, and the perforation of the aorta was on its anterior wall.

The peculiar nature of the pains in this case led Mr. Pakenham to make the diagnosis of aneurism when he first saw the patient. This was the second instance in which, after the publication of Dr. Beatty's case, the disease was accurately diagnosticated from symptoms alone.

The want of accordance between the symptoms and the constitutional state in so many cases of this disease has been already pointed out. In certain instances the fatal rupture may take place without any symptom of constitutional disturbance having occurred. I believe that in such cases the aneurism seldom becomes diffused, and that sudden death is produced by the opening of the sac into a free serous cavity, such as the peritoneum or pleura, or, as in some rare cases, into the stomach or intestines; and the possibility of such an occurrence, even in cases where no tumour has been detected, must never be lost sight of by the practitioner. The two following cases, which were communicated to me by my friend, the late Staff Surgeon Frazer, are highly illustrative of this point:

"A native of India, in the pioneer corps of Ceylon, employed as a labourer in road-making, had an accession of symptoms of severe colic in the year 1823; the same symptoms having several times previously compelled him to abandon his work, and to seek refuge in a hospital. Treatment and short repose enabled him to resume his occupation. On the day of his last admission the urgency of the symptoms was relieved, but on that subsequent an excruciating recurrence of them took place, and in about half an hour ended suddenly in death.

"Dissection, three or four hours after death, showed a great effusion of blood in the belly, the intestines being in fact deluged with the quantity; it proceeded from the bursting of an aneurism, the size of an orange, situated in the abdominal aorta. The real disease in this case was never even suspected.

"A British soldier came into hospital, complaining of violent pain in his back, particularly along his spine, no accompanying morbid signs, either local or constitutional, being indicated. His health was good, the natural and vital functions seemed to be regularly performed: there was no emaciation. Leeches, and I believe some other remedies, were used during his stay in hospital; on his dismissal he acknowledged himself relieved. On attentive consideration of the case, suspicion lurked in the minds of the medical attendants that the case was either feigned or was unimportant; symptoms both local and general being still absent, when he returned a second time, asserting the aggravation of his complaint. After some residence in hospital, he was again discharged without any conclusion having been arrived at as to the nature of the case. The two medical attendants (both experienced in the service) were perplexed, though inclined to believe that the man fabricated much. On his last return to the hospital he was met by one of the above gentlemen, and to the question, what was the matter with him, the invalid replied, 'Oh, sir, I can bear it no longer, I must come to hospital.' In about three or four hours this gentleman was shocked and confounded by hearing of the death of the patient.

"On examination, a large aneurism of the abdominal aorta was found ruptured; the bodies of the adjoining vertebræ were carious. The surgeon of the regiment to which this man belonged was a cautious person, and, fortunately for himself, though he suspected the reality of the disease, avoided those measures which the false conclusion—that the hapless soldier was an impostor—might have led him to adopt."

But where the aneurism, in place of pouring its flood into an unresisting cavity, becomes gradually diffused, or, in other words, where a secondary is added to a primary false aneurism, symptoms more or less characteristic may be observed,—there is often a sudden change in the appearance of the patient which attracts attention. His countenance may become for the first time anxious and sunken, and the pulse small, weak, and accelerated, a change often very striking when its previous regularity is considered. The duration of this state may vary from a period of twenty-four hours to one of several days, during which other phenomena may be produced. These are generally threefold, viz.:—

1. Alterations in the force and sound of the heart.
2. Alterations in the condition of the sac.
3. Evidences of the new sanguineous effusions, or, as we may term them, the secondary false aneurisms.

With respect to the condition of the heart, we find that in most instances its rapidity is augmented and its force greatly diminished. It is often observed to beat with but a single sound, which by some has been described as the first, by others as the second sound. I apprehend that in most instances it is the first sound that is wanting; but this matter requires a more extended observation. I have not met with anæmic murmur of the heart under these circumstances, but doubtless its production is possible.

We have observed two changes in the condition of the sac which have relation to the new effusion of blood. The force of its pulsations may be augmented. This is rare, and probably only occurs at the commencement of the process. It was well marked in Case LXXVI. In most instances the tumour exhibits a feebler pulsation, and becomes less and less defined; so that its discovery is sometimes difficult or impossible. The bellows murmur, so constant an attendant on abdominal aneurism, is lessened in intensity.

Coincident with these changes we observe two classes of phenomena, indicative of the new effusion,—one, the sinking of the powers of life, and that peculiar change in the whole aspect of the case which has been alluded to; and the other, the appearance of tumours either in some portion of the abdomen or in the lumbar regions. In some cases, however, the new tumour or tumours cannot be at first detected. When they present in the lumbar region, an appearance very similar to that of chronic abscess is produced, and the patient may complain of dull pain and weight in the loins, with pain of the side extending to the thigh. In a case in which several tumours were found, pain of a very severe kind preceded the appearance of the tumour.

Dr. Walshe states that the secondary tumours do not pulsate: I have found that the lumbar tumours, and in one case a tumour which formed at the groin, had a feeble and diffused, but still an evident, diastolic pulsation, attended with a soft and equable murmur. In the case of the tumour of the groin the pulsation might have been communicated by the femoral artery, but in the lumbar tumours it was clearly produced in the tumour itself. Pulsation, however, does not always attend these effusions, which, especially when they occupy some portion of the abdomen, often exhibit a solid non-pulsating mass.

CASE LXXVII.—*Aneurism of the Abdominal Aorta; Formation of pulsating Tumours in the left lumbar and iliac regions; Sudden death from effusion of blood into the Pleura.*

A man, aged 43, had suffered from the symptoms of aneurism for three years and a half before his admission into hospital. His disease commenced with a sudden attack of excruciating pain in the lumbar region and abdomen. The abdominal pain, however, soon subsided, but that of the back continued, extending to the left hip. The attack was attended with nausea and thirst. From that time he was subject to frequent invasions of pain, occurring simultaneously in the back and abdomen, and to oppression and sinking about the præcordia, accompanied with a feeling of terror. When admitted, he was emaciated, and found great difficulty in walking, from pain in the left hip and thigh, with occasional spasmodic twitches in the latter. He had severe colic pains referred to the left hypochondrium and iliac fossa, which were relieved by pressure or by lying on the belly. During the exacerbation of the pain the intestines could be felt at the left side contracted and knotty, and severe pain in the back always accompanied the paroxysm. The last three lumbar vertebræ were exquisitely tender, as were also the whole of the left lumbar region and fold of the buttock. The epigastrium and left hypochondrium were full and tender, and on each side the lower ribs were tilted out. The urine was natural, and there was no fever. A pulsating tumour occupied the epigastric region, stretching to the left lumbar region, and attended by a rough bellows murmur.

In the course of a week he was seized with violent colic and intense pain in the back: this attack was preceded by two paroxysms of severe rigor, followed by sweating; it continued during the day, but was relieved by the operation of a draught of castor oil, when he fell into a profound sleep, and awoke free from pain; next morning he had no pain either in back or abdomen, nor was there any tenderness whatever of the tumour. From this time he suffered many agonizing attacks of pain, and was relieved only by opium in large doses, and the application of leeches to the loins; latterly the pain was chiefly referred to the left iliac fossa. In a few days we detected another pulsating tumour immediately within the spinous process of the left ilium; his countenance was sunken; the belly swelled and exquisitely tender; the impulse of the epigastric tumour was decidedly lessened; he complained of numbness in the left leg and hip. Next day the tenderness of the belly was greatly diminished; but from this time he began to sink, frequently falling into a state approaching to syncope. On the fourth day after the appearance of the second tumour he lay down and expired without a struggle.

Dissection.—The body was emaciated; the belly was full, and the epigastric tumour hardly perceptible; but in the left hypochondrium an elongated and fixed tumour extended from an inch above the ilium to the epigastrium. On opening the abdomen, the peritoneum and intestine at first view appeared healthy, but on the left side a remarkable colouring of the parietal portion of the serous membrane was observed, from an extravasation of blood, of not more than a line in thickness, into the sub-serous tissue. On pursuing the examination, two large tumours were found: the first and most anterior, between the abdominal muscles and peritoneum, lying principally on the transversalis, and extending from Poupart's ligament to the last false rib; the second, about half the size, lay on the psoas muscle; these tumours were filled with semi-coagulated dark-coloured blood: there was also effusion under the peritoneum in small quantity, so as to colour the serous membrane of the pelvis and bladder extensively. On raising the sternum, a large quantity of clear, yellow-coloured serum flowed from the left side of the chest. Here upwards of three pounds of blood were found effused, forming a vast coagulum, on removing which, a large fibrinous clot, about the size of the palm of the hand, was seen hanging into the cavity of the pleura, at its diaphragmatic portion close to the spinal column; this proved to be a fragment of the original sac, and immediately above it we found a large opening leading into a cavity situated between the crura of the diaphragm and the dorsal vertebræ. This was the original aneurismal sac. The common iliacs were healthy, but immediately above the bifurcation numerous deposits of white matter were seen under the lining membrane, which in several instances coincided with superficial ulcerations; the whole of the aorta, from the arch downwards, was in this state, no perceptible dilatation of the vessel being observed. Immediately above the cœliac axis the opening into the aneurismal sac, fully an inch in diameter, was found. The sac was of considerable size, its posterior wall being formed by the bodies of the last five dorsal vertebræ, which were deeply eroded. The uppermost cartilages were nearly destroyed, but the most inferior remained unaffected, and formed prominent rings.

The sac had extended under both crura of the diaphragm, under the right being converted into a solid tumour, consisting of concentric layers of fibrine; under the left crus the two openings were evident, one into the left pleura, the other that by which the sub-peritoneal effusion had occurred. The aortic valves were ossified; the heart presented an example of concentric hypertrophy of the left ventricle; the lungs and digestive tube were found to be perfectly healthy.

CASE LXXVIII.—*Aneurism of the Abdominal Aorta; Sub-peritoneal effusion, forming a pulsating Tumour in the left hypochondrium; Extensive separation of the Peritoneum in the lumbar and iliac regions.*

A butcher æt. 34, entered the Meath Hospital in November, 1830, under the care of Dr. Graves. He stated, that about eighteen months previously, while in the act of lifting a weight, he suddenly felt a severe pain in the pit of the stomach, which did not, however, oblige him to quit work; this having continued for a few days, subsided; after which he continued in tolerable health for about twelve months, when after exposure to wet, he was seized with shivering, followed by burning pain and a feeling of pulsation in the epigastrium, vomiting, and palpitation of the heart; since that time he had been affected with lowness of spirits, lassitude, starting from sleep, and troubled dreams, occasional tendency to fainting, great irritability of stomach, and costiveness. About a fortnight before, he had felt pain and a sensation of throbbing in the situation of the left kidney.

On admission he was pale and emaciated, with an anxious countenance, and complained of weakness in the loins and hip, extending down the anterior part of the thigh, accompanied with a sensation of numbness and cold. These parts were not tender on pressure, and he could put his foot firmly to the ground, but was lame, and could walk only for a short distance, the severity of the pain being increased by motion. The pains were much worse at night. A diffused tumour, accompanied with a bellows murmur and pulsating violently, was found occupying the epigastrium, and extending into the left hypochondrium; at the lateral and inferior part of which a somewhat hard and irregular mass, the size of the palm of the hand, could be felt. This mass did not pulsate, but pulsation could be traced along the margin of the ribs, to the situation of the left kidney, where it became very evident. The pulsation in the epigastrium was more evident while he lay on his back; he moaned in his sleep, and often cried, "get off, get off," as if he fancied some person to be lying on his chest. The sounds of the heart were heard over the whole chest. Its impulse was natural. He had no cough or dyspnœa; the bowels were costive, the urinary functions healthy. He used to remark that he felt as if his food stopped at the left kidney.

On the eighteenth day after admission the tumour had extended from the ribs to the crest of the ilium, and to the median line, so that the whole left side of the abdomen was occupied by it. This new deposit did not pulsate; the pulsation in the epigastrium was now confined to a point at the right of the median line, and was greatly diminished in strength.

On the twentieth day his countenance expressed despair, he was deadly pale, and the lips were blanched. The foregoing afternoon he was suddenly seized with intolerable pain in the left groin, causing him to scream with agony, and jump out of bed; the pang continued about five minutes; there was then observed a feebly pulsating tumour in the groin. One hour after the visit he was attacked with intense pain in the left lumbar region, after which he sunk, and died the same day.

Dissection.—Countenance perfectly placid. On opening the abdomen, the muscles and peritoneum, forming the anterior wall, were found in their natural state. The intestines, liver, spleen, and pancreas, were healthy. The serous membrane, covering the lumbar and iliac regions of the left side, was seen pushed upwards and forwards by a considerable quantity of coagulated black blood, which had also displaced the spleen and stomach forward and to the right side. The coagulum extended over the front of the spine, pushing the left kidney considerably forwards and towards the mesial line; the tissue of the left psoas and iliac muscles was infiltrated with blood, which enveloped and pressed on the nerves of the lower extremity of that side. Along the spine the coagulated blood was evidently the result of a more ancient effusion: it had become brown and fibrous, and was disposed in concentric layers, separable by a little force, but connected by numerous reddish fibres.

At the left side of the spine, a little above the kidney, was found the proper aneurismal sac, formed of a dense white cellular tissue, and having a cavity nearly spherical, and capable of containing a small orange. In this sac were two openings: one rather to the left side, through which the larger quantity of blood escaped under the peritoneum; the other communicating with the aorta at its posterior portion, midway between the origins of the cœliac axis and inferior mesenteric arteries. The first, second, and third lumbar vertebræ were destroyed to a considerable extent; but the corresponding intervertebral cartilages were less injured, and their surfaces, though depressed, and as it were, beaten in, were smooth to the touch.

CASE LXXIX.—*Aneurism of the Abdominal Aorta; Neuralgic pain occurring in paroxysms; Effusion of blood beneath the Peritoneum covering the liver and stomach; Sudden death from effusion into the Peritoneum; Absence of erosion of the Vertebræ.*

A middle-aged man had suffered for a year from violent paroxysms of pain in the back, the sides, and the region of the stomach. The pains were not constant, the patient having intervals of complete freedom from suffering. I had no difficulty in recognising an aneurism; for, in addition to the evidences from the symptoms, I found a large pulsating tumour in the epigastrium, having every character of abdominal aneurism, and presenting a distinct bellows murmur. He was admitted into hospital, where, after two days had elapsed, I examined him a second time. To my surprise, the epigastric tumour, which had been so manifest, had disappeared; so that I began to hesitate as to the correctness of the first opinion. The patient now mentioned that when he lay on his side he felt at ease, and the tumour disappeared; but that when he lay on his back the pain returned, and also the tumour. This singular experiment he performed in my presence on several occasions, and always with the above result. I do not remember ever witnessing a more singular circumstance than the re-appearance and, as it were, growth of the tumour under the hand when the patient continued on his back for a period certainly within two minutes. In this case death was sudden. A large clot of blood was found in the peritoneal cavity, proceeding from a rent in the supero-anterior portion of the aneurism. We also found a great effusion of blood confined between the stomach and its peritoneal coat, and a similar effusion had detached a large portion of the peritoneum from the convex surface of the liver. The aneurism lay under the crura of the diaphragm, but there was no erosion of the bodies of the vertebræ.

I shall not here enter into any speculations as to the cause of the disappearance and re-appearance of the tumour according as the patient lay on his side or back; but the fact itself forms an important addition to our knowledge of the signs of abdominal aneurism. The absence of absorption of the vertebræ, notwithstanding that the patient suffered not only from the paroxysmal pains affecting the back, head, and abdomen, but had also constant dull pain in the situation of the aneurism, is of great importance; and the converse fact, which has been more than once observed, of the existence of extensive erosion without these pains, seems to indicate that the pains in abdominal aneurism are more dependent upon its action on soft parts than upon the structures of the spine; indeed, I have a strong impression that the absorption of the bones and intervertebral cartilages is a painless process.

In a case observed by Dr. Hutton the tumour reached from the crest of the ilium to the inferior margin of the scapula. Much of this enlargement took place in a sudden manner, the patient complaining of a sense of faintness. At that time a bellows murmur which had been distinctly heard along the spine became diminished, and the volume of the pulse in the left femoral artery was lessened.[1] The patient died from syncope, having been previously jaundiced. Several vertebræ were deeply eroded, and the spine was curved both laterally and posteriorly, and yet the peculiar neuralgic pains were not complained of.

In a remarkable case by Dr. Law the diffusion of the aneurism was marked by a protracted tremor or rigor; soon after which it was found that the pulsations of the original tumour, as well as the murmur which attended them, had disappeared. The left portion of the abdomen had become tender to pressure. Part of it was distended, giving a clear sound, while the remainder was dull on percussion, and in the course of some hours the pulse at the wrist disappeared. On the third day a diffused bellows murmur could be heard in the epigastrium; the heart's action was rapid, and the sounds single. There was considerable fulness between the spine of the left ilium and the symphysis pubis. The patient expired suddenly on the fourth day after the rigor. At a level with the cœliac axis the aorta was found dilated, and communicating with a bilocular cavity; one portion of which was capable of containing an orange, its posterior wall being formed by the eroded bodies of the vertebræ. The larger cavity lay on the left side of the spine. It contained a mass of coagulated blood, weighing at least two pounds, and extending from the diaphragm to the brim of the pelvis. This appeared to have opened suddenly into the cavity of the pleura, which contained not less than three pounds of blood.

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[1] This case was at first under the care of Dr. Monahan, who recognised the disease. The patient was afterwards treated by another practitioner for renal disease.—(See Transactions of the Pathological Society of Dublin, November 26, 1842.)

In the cases which we have now been considering there was no difficulty in determining the nature of the secondary tumours, inasmuch as the existence of an abdominal aneurism in its usual form had in some cases been recognised before the new tumours were established, and in others continued to exhibit its characters, though in a manner more or less modified. We have now, however, to examine a case in which the existence of the secondary effusions caused the greatest difficulty in diagnosis—the question being, whether a certain abdominal tumour was really aneurism, or whether it was a solid tumour, probably malignant, which received a communicated pulsation from the abdominal aorta.

CASE LXXX.—*Vast false Aneurism of the Abdominal Aorta; Secondary effusion of blood into the transverse mesocolon; Severe neuralgic pains aggravated by exertion; Absence of erosion of the Vertebræ.*

A carpenter, aged 53, of intemperate habits, entered the Meath Hospital in January, 1853. A large tumour, lobulated, irregular in form, and apparently solid, occupied the centre of the abdomen. It bore the closest resemblance to one of the irregular encephaloid tumours of the belly, and extended from the epigastrium to a little below the umbilicus, sending also a prolongation into the left hypochondrium.

The appearance of the patient was expressive of abdominal disease and of continued constitutional suffering. He vomited his food soon after taking it. This symptom had existed for the last six months, the entire duration of the disease being nine months. He also complained of severe pain in the back, which had existed from the first invasion of the symptoms.

Until the last nine months this patient had enjoyed good health. His first symptom was the appearance, without any previous indisposition, of a small circular tumour in the epigastric region, which after a few days began to pulsate. As it increased he felt a severe pain, most acute in the back, and radiating downwards into the abdomen. This was aggravated by exertion. The irritability of the stomach was established at an early period of the disease: he, however, continued at his trade to within ten days previous to his admission.

Excepting the irritability of the stomach, the symptoms resembled those of aneurism, and the opinion that such was the nature of the disease was strengthened by the fact that the tumour had a strong and extended pulsation; yet I felt extreme difficulty in making up my mind as to the nature of the disease.

In its form the tumour closely resembled a cancerous mass—presenting the irregularity of shape, the lobulated protuberances, and the varying resistance to pressure at different points, so commonly seen in that disease. Again, the pulsations, though diastolic, were peculiar. The force of the diastolic throb was by no means commensurate with the size of the tumour, and might well be supposed to correspond with that of the throbbing of a cancerous mass of various degrees of consistence, lying above the aorta. But further, the whole mass appeared to be thrown upwards at each pulsation, just as may be seen in tumours of the abdomen. Lastly, two distinct kinds of bellows murmur were detected. One, soft and diffused, existed in the upper or epigastric portion of the tumour; the other, sharp and almost musical, was confined wholly to that process of the tumour which extended into the left hypochondrium, and was the size of a hen's egg. All these circumstances, except, perhaps, the last, were in favour of the diagnosis of organic tumour; yet we had met with no case either of aneurism or of cancer presenting two forms of bellows murmur so distinctly localized as in this instance.

For several days I strongly inclined to the diagnosis of a cancerous or solid tumour of some kind, when it was discovered that the bellows murmur at the epigastrium could be traced upwards, becoming more and more intense towards the arch of the aorta. There was no murmur in the heart, and the discovery, which was made by one of our clinical students, led me to doubt greatly the correctness of my first impressions. Three days before the patient's death he suddenly became greatly prostrated: his death was easy, though sudden. The heart and its valves were found perfectly healthy. Some patches of atheroma existed in the arch of the aorta, but its remaining portion was healthy as far as the cœliac axis, below which was an oval opening in the vessel not less than 2½ inches in length, which communicated with a vast aneurismal cavity, under which was seen a second tumour of the colour of blood, lying across the abdomen and stretching into the left iliac fossa. It was like a pillow of blood, and was formed by a vast effusion, proceeding from a large rent in the lower portion of the sac, by which the blood was poured between the layers of the mesentery. The appearance of the colon, which was contracted and pale, was very striking, as it formed a fringe or border a little above the lower edge of the tumour. The cavity of the peritoneum did not contain a drop of blood, and the bodies of the vertebræ were free from erosion.

There is good reason for believing that in this case two forms of pulsation affected the tumour—one, the true diastolic, belonging to the aneurismal sac; and the other, the communicated throbbing, which was felt over the great mass of coagulated blood in the folds of the mesentery. In distinguishing aneurism from tumours of a different nature, we have hitherto placed great reliance on the character of the pulsation; but it must henceforward be borne in mind that not only the diastolic, but that simpler pulsation in which the mass is thrown upwards and forwards at each stroke of the artery, may occur in the one case, and yet the disease be aneurism.[1]

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[1] The following observations are extracted from the report of a clinical lecture which was given in the Meath Hospital a short time before this patient's death. I had been observing on the circumstances which at first inclined me to reject the opinion that the disease was aneurism:—

But other conditions besides these bloody tumours obscure the diagnosis of abdominal aneurism: of these, the displacement of the solid viscera is especially to be noticed. We have seen that even the heart may be displaced: but the organs most com-

"The patient has a loud bellows murmur in his thoracic aorta, which I was not until this day aware of. It is loudest at the arch, and he has it in the course of the descending aorta. Now, this murmur either proceeds from a general condition of anæmia, or from disease of the aorta. There is nothing more rare than an anæmic murmur in a man of this class. It is extremely rare to find an anæmic murmur in the aorta and not in the heart; he has no murmur in his heart. Therefore, there is the greatest probability that this murmur in the aorta proceeds from organic disease; that it is an organic murmur, to use the phrase now adopted. It might be supposed that it was propagated upwards from the abdominal aorta. On that subject all we know is this, that in a large proportion of cases of abdominal aneurism which present murmur, where the heart and the thoracic aorta are healthy, there is no murmur transmitted from the abdominal upwards to the thoracic aorta. This is a curious and important fact, and, indeed, so far as we know, retrograde murmurs are extremely rare. The chances, then, are, that he has disease of the aorta; and if he has organic disease of the thoracic aorta, it greatly increases the probability that any suspicious circumstances in connexion with the abdominal aorta are indicative of disease there also. One point more:—There are two kinds of tumours, which, when lying upon arteries, receive pulsations: one the solid, the other the semi-fluid tumours. Now, if this case be not aneurism, it must be one of those semi-fluid tumours. I showed, many years since, that semi-fluid tumours within the cavity of the chest might simulate aneurism: that they might have a diastolic pulsation; and present a bellows murmur. I need not say that they also present those other phenomena of aneurism which proceed from the displacement of surrounding parts. But as far as I know of cases of semi-fluid tumours which pulsate and have murmurs, owing to the pressure upon an artery, the murmur is equably diffused over them. We have not a loud murmur in one part of the tumour, and a feeble murmur in another; nor have we, as far as I know,—and we must only argue from what we do know,—a murmur of one tone at one part of the tumour, and a murmur of another tone at another part. We have these things in aneurisms, but we have them not in semi-fluid tumours lying upon the aorta, so that, under these circumstances, considering the great difficulty of the general diagnosis,—considering that the man has certainly disease of his aorta,—considering that the tumour presents more or less of a diastolic pulsation, and that it has two kinds of murmurs in it, differing in intensity, and differing in tone,—we have enough, at all events, to warrant us—I will not say in declaring that the case is aneurism—but in doubting that it is one of cancer. And in the present state of our knowledge, I think this is a case in which, having balanced all the points that you can arrive at, we must remain in some doubt as to the true diagnosis. Fortunately, it signifies nothing as to the safety or to the treatment of the patient, whether the disease be an aneurism or a cancer. The only possible utility in the monly disturbed, and thrown out of their natural position, are the spleen, the kidney,—in most cases the left kidney,—and lastly, the liver. In such cases, supposing the aneurism not to have become diffused, solid masses may be presented, which only receive the communicated or non-diastolic pulsation; and in this way difficulties similar to those which occurred in the instance now recorded will have to be encountered. In the case by Dr. Beatty the protrusion downwards and forwards of the liver towards the close of the disease, and its return to its natural situation after the rupture of the aneurism, are matters of great interest. A somewhat similar occurrence was observed by me in a case of aneurism of the hepatic artery, which occurred in the portion of the vessel covered by the capsule of Glisson. Death was caused by sudden rupture into the peritoneum. The liver was found to be smaller than natural, and the gall-bladder and hepatic ducts were distended to the last degree by bile. The patient had persistent jaundice. In this case no pulsation had been observed during life.[1]

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differential diagnosis might be where the question was as to the probability of sudden termination of the patient's existence, if such a question as that was of importance. It might be important if the person had weighty affairs to settle, and that it was a great object for him to live for a certain period of time, and so on. But such a case is not very common; and as far as the practice of medicine is concerned,—as far as the safety and well-being of the patient are concerned,—it fortunately signifies nothing. The imperfection of our knowledge of diagnosis is a matter of no consequence to him. It might be of importance to us if we were vain of our diagnostic powers,—if we were not humble enough to say that we will not venture upon a differential diagnosis here;—I do not venture upon a positive diagnosis in this case. Such a case is, fortunately, of rare occurrence. Here again we have an illustration, in chronic disease, of the principle which I illustrated to you with respect to acute disease,—that where differential diagnosis is difficult or impossible, it is in practice often unnecessary, so far as immediate action is concerned."

[1] See Dublin Medical Journal, First Series, vol. v., p. 401. My friend, Dr. Gairdner, in a report of a case of aneurism of the superior mesenteric artery (Edinburgh Monthly Journal, 1850,) has quoted this case. It is to be observed, that one of the first symptoms was an attack of hæmatemesis, which, in Dr. Gairdner's opinion, indicated that a direct passage of the blood from the aneurismal sac into the hepatic ducts had at one time occurred. He suggests that, had a more careful dissection been made, the traces of such a communication would have been discovered. Without impugning the general doctrine in question, I would suggest, that in dealing with or drawing conclusions from other

It is much to be doubted whether we have as yet any diagnostic of absorption of the bodies of the vertebræ. An examination of the cases which have now been given will show that the lesion is by no means constant. In certain cases it is true that tenderness of the dorsal spines may be observed; but this is insufficient to prove the existence of a painful caries. When it is recollected that on the one hand dreadful neuralgic pains have occurred without any absorption of the bones, and that on the other this lesion has been present without the existence either of the lancinating pains or the dull, constant, boring sensation, as described by Dr. Law, we have strong reasons for believing, as has been already stated, that the absorption of the bones is a painless process. The pains in question appear to proceed, on the one hand, from some inflammatory or neuralgic condition of the sac; and on the other, from its effects on surrounding nervous filaments: and it may be stated generally, that the higher up is the perforation of the artery, the greater is the probability that the disease will be attended with severe pain. The keenest suffering has been observed when the aneurism lay between the crura of the diaphragm, or when the opening into the artery was but a little below the cœliac axis. It has been suggested that when the opening has been on the anterior wall of the artery the disease is attended with less suffering than in the opposite case. Upon this question my experience does not warrant me in pronouncing any opinion; but I believe that aneurism from an opening in the anterior wall of the artery is rarely to be met with.

In connexion with this subject, the following case possesses great interest:—A professional friend of mine was consulted by a gentleman who laboured under symptoms characteristic of abdominal aneurism. There was a deep-seated bellows murmur in the left epigastric region; and, after a careful examination, it was determined that abdominal aneurism existed. The nature of the case was explained to the friends of the patient, and rest, anodynes, and a nutritious but moderate diet were prescribed. The

men's labours, it is a safer course to take them as they stand than to assume error or deficiency on the part of the observers. There is no reason to suppose that any such communication ever existed. Great care was taken in the dissection, in which I was aided by Professor Porter and the late Dr. Houston.

vever, became dissatisfied. He was an ardent sports- nged to resume his favourite pastime of fox-hunting. actitioner was consulted, who came to the conclusion ease was simply neuralgic, and advised the patient to d, to mount his horse, and to join the hounds on the meeting. This advice was followed; and it is a re- act that during the next week or ten days this gentleman went out hunting, and, though exposed to great ex- ent and fatigue, experienced a total exemption from all his oms. The pains, however, s n returned, the aneurismal r increased, and he agai d himself under the care of mer physician. His death was sudden; and on dissection as discovered that three or four of the dorsal vertebræ were extensively eroded. This case, while it exemplifies the effect mental impression and pleasur le excitement in suspending pains of abdominal aneurism, as was observed by Dr. Beatty, goes to prove the non-inflammatory nature of the absorption the bones; and it shows what could not have been anticipated, t a patient labouring under aneurism, and also extensive absorption of the bodies of the vertebræ, may be exposed to extraordinary exertion, not only without pain, but with actual, though temporary relief of his sufferings.

DISEASES WHICH SIMULATE ABDOMINAL ANEURISM.

The affections which simulate abdominal aneurism, or which at least are often mistaken for it, may be divided into two classes: viz., those in which tumours of the belly receive a communicated pulsation, and those where there is simply an increased action of the abdominal aorta. Of the first class, hypertrophy of the liver, especially of its left lobe; pyloric and pancreatic tumours; and lastly, large tumours in the mesentery, generally cancerous, are examples. In the second category we find cases of merely nervous throbbings of the abdominal aorta, or of increased action of the vessel, symptomatic of irritation in some portion of the intestinal mucous membrane or glands.

With respect to the first class, we find that solid tumours, such as cancer of the pylorus or hypertrophy of the left lobe of the liver, are less liable to be mistaken for aneurism than those of which the structure is softer or semi-fluid, as in cases of encephaloid tumour; for although in all these instances there is the communicated vertical throb, yet in the latter, from the nature of their structure, the impulse becomes more or less diastolic.

When we recollect that in these cases three of the most important signs of aneurism are often present, namely, tumour, pulsation, and bellows murmur, it is natural that they should be mistaken by inexperienced men; and we have seen that there are cases in which it is very difficult to settle the question. When the tumour is solid there is generally less difficulty. The pulsation may often be determined to be undiastolic. The tumour is sometimes movable, so that we can at will cause the pulsation to appear and disappear, according as the mass is brought to bear on the vessel, and the murmur, resulting as it does from pressure on the aorta, may be made to vary in intensity, or even to disappear, by moving the tumour to either side, but most often to the right. To these points we shall return.

Pyloric tumours, some floating tumours in the mesentery, and certain instances of hypertrophy of the left lobe of the liver, may exhibit these conditions. On the other hand, tumours which are fixed, and especially when they are of a soft consistence, either in part or throughout, cause greater difficulty, for then we cannot obtain the decisive evidence of the arrest of pulsation by the displacement of the tumour. And again, the nature of their contents makes the pulsation in a degree diastolic. In dealing with these cases many points must be considered. First of all, the history of the disease and the constitutional state of the patient claim attention; and we are to remember that in many cases of abdominal aneurism, where there has been as yet no secondary hemorrhage, no diffusion of the aneurism, the constitutional state may remain good, even though there have been severe neuralgic suffering. The existence, then, of constitutional disturbance, especially if this date from a time anterior to the appearance of the tumour, or have attended it in its early periods, is an argument, so far, against its aneurismal nature. We must inquire as to the occurrence of general debility, of loss of flesh and appetite, of a cachectic state, or of fever,—inflammatory, hectic, or irritative. There is nothing more remarkable than the rarity of any form of fever in connexion with aneurism; and further, it happens in most cases that the first appearance of the disease has not been preceded by evidence of deranged health.

We are next to consider the position of the tumour. In most cases which I have seen, where an abdominal aneurism existed, the tumour first presented high up in the belly, and to the left side. The most usual situation is the left epigastric region, the tumour extending under the false ribs. I only remember one case, in which the tumour presented in the first instance in the centre of the epigastrium. Again, the most frequent seat of the perforation is at or immediately below the cœliac axis, and hence it may be concluded that the early appearance of a pulsating mass in the mesian line at a point below the epigastric region is more indicative of external tumour than of arterial disease. In cases where the tumour has begun inferiorly and extended upwards, there is the greatest probability that the disease is not aneurism.

Proceeding in his investigation, the observer will now examine as to the fixity or movability of the tumour. I believe that the tumour formed by a non-diffused false aneurism of the aorta is immovable. Aneurisms of the second or third order of vessels might be movable; and I think that I once saw a case of this sort. But these instances are so rare that in practice their consideration may be omitted. On the other hand, those tumours which receive a communicated pulsation are commonly more or less movable by the hand in the transverse or vertical direction, and in some, a forced action of the diaphragm causes a gliding motion downwards.

The feel of the non-aneurismal tumours is generally characteristic. It conveys the impression of hardness, and more or less irregularity in form. So long as an aneurism has not become diffused, this result will be useful in diagnosis, as in most cases of the disease the sac can be felt as a deep-seated, rounded, and immovable tumour, which, though sometimes hard, is generally not so resistant to pressure.

Continuing the examination, we are next to look for three remarkable conditions, none of which I have ever met with in aneurism of the abdomen. These are:

1. A varicose state of the epigastric veins.
2. The existence of ascites.

3. Effusion of lymph, giving the peritoneal friction signs.

The frequent occurrence of these conditions in organic tumours of the belly, especially those of a malignant nature, and their absence, or at all events their extreme rarity, in aneurism, furnish us with valuable diagnostics.

In examining for the presence of serous effusion, the amount of which is often but small, the patient should be placed in the upright position, a little inclined forwards, and thus an effusion, which would escape examination in the recumbent position, will give a distinct fluctuation in the lower part of the belly.

The last subjects for investigation are the characters of the bellows murmur, and the force and nature of the pulsation. We cannot as yet say that the murmur so often produced in a solid tumour differs by any merely acoustic character from that of an aneurism. As a general rule it is louder at a point lower down than that in which the aneurismal murmur is commonly developed. Where the tumour can be moved transversely, so as to relieve the vessel from pressure, we can sometimes cause diminution or extinction of the murmur; and I have sometimes succeeded, by placing the patient on his hands and knees, in removing all murmur, and even pulsation, from the mass.[1] So far as I know, these communicated murmurs are found to present the same character at different portions of the tumour, although they may be more distinct at one point than another. It is to be remembered that the reverse of this occurred in the case of effusion into the mesocolon. Here there were two distinct forms of bellows murmur, and, reasoning from what I have observed of diffused aneurismal tumours in the loins, I cannot doubt that one of these murmurs belonged to the secondary, and the other to the primary, false aneurism.

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[1] In a paper on the diagnosis of aortic aneurism, Dublin Journal of Medical Science, vol. ii., Dr. Corrigan has recorded a case of abdominal aneurism, in which bellows murmur, though absent in the erect, became manifest in the horizontal position. He conceives that, in the earlier periods of the disease, the hydrostatic pressure from above, by keeping the sac in a state of tension, prevents the occurrence of bellows murmur; but that it may be produced in the recumbent position in consequence of the relief from pressure having lessened the tension of the sac. In this case about three weeks elapsed before any distinct tumour could be perceived. "Sometimes," he observes, "a moment's lying in the horizontal position has been sufficient to make the *bruit de soufflet* evident; at other times it required a lapse of two or three minutes before the sound became very distinct." Dr. Corrigan properly observes that this variation of the murmur on position might be expected in cases of tumours lying on the aorta, but he thinks that in most instances the murmur would be propagated along the course of the vessel. I apprehend that the occurrence in question will not be found a satisfactory diagnostic between aneurism and other tumours of the belly, but it is in cases where the existence of any tumour is doubtful that Dr. Corrigan's observations will be found valuable. In Dr. Lees' case the murmur disappeared in the erect position.

The force of the pulsation is next to be considered; and it may be stated generally that these communicated pulsations are often even more manifest than those of abdominal aneurism itself. In the latter case the tumour is generally deep-seated and high up in the abdomen or under the left ribs; and it is only by a forcible pressure inwards that we can perceive the characteristic power of the aneurismal throb. But in the former a large tumour often exists lower down, and in direct contact with the anterior parietes of the belly, and as this is thrown upwards and forwards at each stroke of the artery, the pulsation is manifest at once to the eye and hand.

It has been already shown that an accidental increase of the throbbing of the abdominal aorta in a case of permanent patency of the aortic valves is liable to be mistaken for aneurism. I am persuaded that this error is frequently committed. The practitioner should never omit to examine the heart carefully, even in cases of abdominal aneurism; and I have given an instance in which this negligence on my own part had nearly led to an error in diagnosis. We have seen that throbbing of the upper portion of the thorax, taken alone, is of little value as indicative of aneurism, when from deficient aortic valves, and hypertrophy of the heart, the action of the whole arterial system becomes more apparent. The same rule would apply to the case now before us; and though the combination of abdominal aneurism and permanent patency of the aortic valves is extremely rare, it is plain that we should be slower in setting down the throbbing of the abdominal vessels as aneurismal, when there is evidence of deficient aortic opening.

Finally, it has been shown, that when the tumour is of a semi-fluid consistence, the pulsation may be to a certain degree diastolic. I only once observed the occurrence of complete and vehement diastolic pulsation in a non-aneurismal tumour of the belly. I was brought by Dr. Macready to see a young man, the history of whose case was as follows:—

He had always enjoyed good health up to the period of his attack. After exposure to cold he was seized with a rigor, followed by high inflammatory fever and intolerable pain in the back. These symptoms having continued for some days, the following changes took place. The pain of the back ceased, and he complained of extraordinary distress and suffering in the right epigastric region. The fever changed its type, and resembled the most violent hectic, attended with copious sweatings, and in a short time a pulsating tumour appeared in the upper portion of the belly. When I saw the patient, the pulsations were completely diastolic, and indescribably vehement. We came to the conclusion that the disease was probably hepatitis, tending to abscess, and this opinion was strengthened by our discovering that an intense peritoneal friction, evident to the hand and ear, was produced over the tumour whenever the patient drew a deep breath. In a few days he discharged, both by vomiting and stool, a vast quantity of purulent matter: his recovery was perfect.

This case and others seem to furnish another differential diagnostic between the simulated and real aneurisms of the abdominal aorta. Dr. Beatty has shown that peritoneal friction is commonly developed in organic tumours of the belly. I have never found it in any case of abdominal aneurism, even where rupture of the sac and effusion had taken place:—so that the absence of the physical signs of serous inflammation may give us a diagnostic which will be occasionally useful in distinguishing aneurism from those tumours by which it is simulated.

Sympathetic or nervous throbbings of the abdominal aorta, independent of organic change in any part of the circulating system, are seldom likely to be mistaken for abdominal aneurism if the observer take in all the circumstances of the case. The following examples of the affection are not uncommon, the symptom being presented in various degrees of intensity:—

1. Hysteric or nervous throbbing.
2. Dyspeptic pulsations.
3. Pulsations symptomatic of irritation in some portion of the digestive tube, often attended with fever.

4. Increased action preceding the appearance of the catamenia.

5. Pulsations occurring in the early and middle periods of pregnancy.

Of these the first two most often mislead the inexperienced observer. The coexistence of fever in the third case is in itself nearly conclusive against aneurism.

To all these cases the same rules of diagnosis will apply. With respect to the mere nature of the throbbings I have little to add to the description given by Hope; but it is easier to learn their characters at the bedside than to describe them. The throb is generally jerking and sudden: it is rarely diastolic, like that of an aneurismal tumour, but strikes upwards as the patient lies on the back, and if diastolic, not to an extent greater than the calibre of the artery. It is not a circumscribed, but rather an elongated pulsation, sometimes occupying the whole line of the vessel to its bifurcation; and in many cases it differs from the throbbings of an aneurism in this, that its intensity increases from above downwards, and has its maximum at the umbilical region, and that its force and character are continually varying.

But the great source of diagnosis is from considering the age, sex, and habit of the patient, the mode of invasion of the symptom, the antecedents of the case, and the want of the symptoms of abdominal aneurism.[1]

TREATMENT OF ABDOMINAL ANEURISM.

On this subject it is unnecessary to dwell at any length, for it is obvious that the same general principles which apply to the treatment of thoracic aneurism will hold equally good in the abdominal cases. For the relief of the neuralgic suffering our great resource is opium, in its various modes of administration. Other narcotics may fairly be employed, and it is probable that where there is no evidence of a weakened state of the heart, we might employ chloroform with good results. Should this be found to relieve the torments of abdominal aneurism, another leaf will be added to the civic crown which Dr. Simpson has earned so well. The use or omission of aperient medicine must be entirely regulated by the circumstances of each particular case. With respect to local bleeding, I do not think that its employment is followed by the same beneficial effects as in thoracic aneurism; I have used it, however, in but few cases.

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[1] The abdominal throbbing which appears symptomatic of actual irritation or inflammation in the intestinal tract, and probably analogous to that of the carotid in cerebritis, or of the radials in whitlow, was described by me in my Researches on the Diagnosis of Aneurism, Dublin Journal of Medical Science, vol. v. The existence of this form has been acknowledged by Dr. Hope.

RECAPITULATION.

1. That aneurism of the abdominal aorta, especially when it arrives high up in the course of the vessel, may be attended with severe and peculiar neuralgic pains.

2. That in certain cases these pains are so characteristic as to enable us to diagnosticate the disease with probable accuracy.

3. That they may exist for a long period without any symptom of constitutional ailment.

4. That they cannot be attributed solely to erosion of the vertebræ.

5. That aneurism in the abdomen is less often connected with extensive arterial lesion than when it occurs in the thorax.

6. That the want of proportion between the intensity of the sufferings and the amount of constitutional disturbance is often an important source of diagnosis.

7. That although the occurrence of constitutional suffering is generally seen when the aneurism becomes diffused, yet that in some cases it seems to attend the earlier stages of the disease.

8. That fever is absent in the earlier stages.

9. That fever is rarely met with in any stage of abdominal aneurism. It is absent in the first stages before the aneurism becomes diffused; and when appearing after that change, it is of a low, irritative type.

10. That the pulsations in the disease are generally single, and that they are more certainly attended with bellows murmur than those which accompany thoracic aneurism.

11. That displacement of important organs may long precede the fatal rupture or the diffusion of the aneurism.

12. That the pulsations of the tumour are in most cases single, but that in a case where the aneurism was seated high up, a double pulsation has been observed.

13. That abdominal aneurism may cause displacement of the heart to the right side.

14. That the murmur has been known to disappear in the erect position.

15. That true aneurism of the abdominal aorta is rarely met with.

16. That death may occur from the rupture of the original sac into the peritoneal or pleural cavity; by the aneurism becoming diffused (secondary false aneurism;) or by simple exhaustion of the patient, without rupture of the sac.

17. That the patient often obtains relief by lying on his face; in one case all signs of the tumour disappeared on turning to the left side.

18. That sudden and fatal hemorrhage may proceed from the opening of the diffused aneurism into a serous sac.

19. That the diffusion of the aneurism is often indicated, not only by a remarkable change in the constitutional state, but by alterations of the physical phenomena of the sac and of the heart.

20. That great tenderness of the belly may be thus produced, and yet no peritoneal inflammation exist.

21. That local suffering may attend the occurrence of the effusion.

22. That the new deposits of blood may present a feeble diastolic pulsation and a soft and diffused bellows murmur; in some cases, however, they appear as non-pulsating masses, or receive a communicated stroke from the subjacent or contiguous artery.

23. That, as a general rule, we find the force of the heart, and that of the throbbings in the original sac, diminish when the aneurism becomes diffused. In one case, however, excitement, both of the heart and aneurism, preceded the accident.

24. That in cases where much blood has been effused, the sounds of the heart may become single, the original tumour less distinct, and the bellows murmur diminished.

25. That in these instances it appears probable that it is the systolic sound of the heart that is wanting.

26. That in a case where a copious effusion had occurred between the folds of the mesentery, two murmurs, differing in seat, tone, and quality, were observed.

27. That all the solid viscera of the abdomen are liable to be displaced in the course of the disease.

28. That the violent neuralgic pains are not necessarily connected with absorption of the vertebræ; and that this lesion may be found without even paroxysmal or constant pain.

29. That two forms of pain are often met with,—one, violent, neuralgic, and paroxysmal; the other, more dull, deep-seated, and boring.

30. That the tumour formed by a non-diffused aneurism is immovable.

31. That the first development of a murmur low down in the abdomen should incline us against the diagnosis of aneurism.

32. That the abdominal tumours which most simulate aneurism are those whose consistence is semi-fluid.

33. That when these tumours are movable, the pulsation, and even the murmur, can be made to appear and disappear.

34. That while the progress of an aneurismal tumour is generally from above downwards, that of the solid tumours is more often from below upwards.

35. That the first appearance of pulsation, at some point low down in the belly, indicates that it is communicated to, rather than inherent in, the tumour.

36. That there are three important conditions occasionally attendant upon solid tumours of the belly which we have never observed in aneurism. These are:—

a. Collateral venous circulation, as shown by the enlargement of the epigastric veins.

b. The existence of ascites.

c. The occurrence of friction sound and vibration over the tumour.

37. That in cases of permanent patency of the aortic valves, a temporary increase of the pulsations of the abdominal aorta, probably induced by sympathetic irritation, has led to the erroneous diagnosis of abdominal aneurism.

38. That a tumour with fluid contents, such as an hepatic abscess, may have a completely diastolic pulsation. Here the diagnosis will depend on the preceding and accompanying circumstances of the case.

TABLE OF CASES.

PERICARDITIS.

Coexistence of friction sounds with vast effusion of liquid into the pericardial sac; dissection.—Dr. Corrigan. *Abstract.* 86.
Pneumo-pericarditis. *Abstract.* 37.
Pneumo-pericarditis from fistulous opening into the sac; dissection.—Dr. Graves. *Abstract.* 89.
Similar case; dissection.—Dr. M'Dowell. *Abstract.* 41.
Pericardial effusion without formation of false membrane or friction sound; dissection. Dr. Mayne. *Referred to,* 48, *note.*
Pericardial effusion with sound of mill-wheel; dissection.—Bricheteau. *Abstract.* 44, *note.*
Pericarditis, with sounds of heart striking against the pleura; dissection.—*Abstract.* 45.
Pericarditis, with triple friction sound. *Abstract.* 45.
Pericarditis, with hypertrophy and dilatation, accompanied by two loud prolonged sounds of different tones; dissection.—Dr. Graves. *Abstract.* 47.
Pericarditis, with manifest friction vibration, in which no friction sounds were detected until the day before death; dissection.—*Abstract.* 48.
Two cases in which no murmur preceded the attrition sounds.—Dr. Mayne. *Referred to,* 50, *note.*
Case in which the distention of the pericardium reached to the first rib.—Dr. Corrigan. *Referred to,* 58.
Epigastric tumour from distention of the pericardium.—Corvisart. *Referred to,* 58.
Dilatation of the præcordial region from the same; dissection.—Louis. *Abstract.* 59.
Extrusion of the left lung upwards, from pericarditis, with extensive effusion; dissection.—Dr. Graves. *Abstract.* 59.
Upward displacement of left lung from complication of pericardial with pleuritic effusion. *Abstract.* 60. Referred to as unattended with dysphagia. 73.
Rheumatic fever, in which pericarditis preceded the inflammation of the joints. *Abstract.* 62.
Similar case.—Dr. Graves. *Abstract.* 62.
Pericarditis, with exacerbations resembling those of angina pectoris; dissection.—Andral. *Abstract.* 5.
Cases from Testa, of dysphagia attending pericarditis; dissection in three of them. *Abstracts.* 70.
Dysphagia, accompanying pleuritis, with effusion. *Abstract.* 71.
Aphonia accompanying pneumonia. *Abstract.* 72.
Changes of tone of voice accompanying pericarditis. *Abstract.* 72.
Case of the courier in Morgagni. 73, *note.*
Pericarditis, accompanied with sudden destruction of the right eye; dissection.—Corvisart. *Abstract.* 74.
Pericarditis, attended with ecchymosis and inflammation of the right eye.—Corvisart. *Abstract.* 74.
Acute dry pericarditis following the disappearance of a cutaneous disease; production of the leather-creak sound within a short time before death: dissection. 76.
Acute dry pericarditis, with hypertrophy and dilatation of the heart; dissection.—77.
Pericarditis in a child of four months; dissection.—Dr. Lees. *Abstract.* 77, *note.*
Inflammatory effusion into pericardium without development of friction sound; dissection.—Dr. Mayne. *Abstract.* 79.

Another case of the same; dissection.—Dr. Mayne. *Abstract.* 79.
Acute pericarditis with pneumonia and arthritis. 80.
Acute arthritis; pericarditis; double pleuro-pneumonia; recovery. 83.
Pericarditis supervening on acute empyema of the right side; protrusion of the diaphragm, and displacement of the liver; dissection. 86.
Extensive empyema of the left pleura; dexiocardia; acute latent pericarditis; intense friction sound, disappearing with a nearly complete obliteration of the pericardial sac; dissection. 89.
Chronic empyema of the left pleura; intercurrent latent pericarditis affecting the displaced heart. 92.
Acute gangrenous abscess of the lung; pericarditis; dissection. 93.
Pericarditis supervening during the last month of phthisis.—Louis. *Abstract.* 94, *note.*
Small-pox with pericarditis; dissection.—Andral. *Abstract.* 95.
Pericarditis combined with aneurism of the aorta; dissection.—Sir Philip Crampton. *Abstract.* 95.
Pericarditis from discharge of small shot from a gun. *Abstract.* 97.
Two attacks of rheumatic carditis within a period of seven months, with an intervening seizure of apparently nervous palpitation; use of wine; recovery. 104.
Paracentesis in dropsy of the pericardium.—Dr. Schuh. *Abstract.* 107, *note.*
Two cases of the same operation.—Dr. Karnwagen. *Referred to,* 107, *note.*

ENDOCARDITIS AND MYOCARDITIS.

Symptoms of carditis, valvular murmur being only occasionally developed; absence of friction signs; death. 120.
Inflammation of pulmonary valves, unattended by murmur, with softened heart; dissection.—Dr. Graves. *Abstract.* 122.
Dilatation and hypertrophy of the heart; ossification of the mitral valves unattended by murmur; supervention of acute endocarditis, developing a loud murmur with the first sound; dissection. 122.
Myocarditis; dissection. *Abstract.* 125.
Gangrene of the heart; dissection.—Testa. *Abstract.* 126.
Ulceration of left ventricle.—Testa. *Abstract.* 127.
Chronic ulcerative endo-myocarditis, with aortitis, rupture of the heart; dissection.—Testa. *Abstract.* 127.
Hypertrophy of heart with valvular disease; abscess of heart.—Dr. Graves. *Abstract.* 128.
Arteritis of right common iliac artery, its divisions and branches, and resulting in polypus occupying these vessels; dissection.—Drs. Graves and Stokes. *Abstract.* 131.
Symptoms of acute endocarditis; doubling of the second sound. 132.
Rheumatic endocarditis; distinct doubling of the second sound. 133.
Arthritis; cardiac complication; bellows murmur accompanying the first sound; doubling of the second sound while the patient remained in the horizontal position. 134.
Doubling of one of the sounds of the heart in peripneumonia notha. *Alluded to,* 135.
Purulent cysts in both ventricles; protracted symptoms of phlebitic disease; dissection. 137.
Purulent cysts of the heart; dissection.—Mr. O'Ferrall. *Abstract.* 138.
Purulent cysts of the heart; dissection.—Mr. O'Ferrall. *Abstract.* 139.
Coagulum in left ventricle and aorta; malignant cholera; dissection.—*Abstract.* 140.
Purulent cysts of heart; phthisis pulmonalis; dissection.—Dr. Bigger. *Abstract.* 141.

DISEASES OF THE VALVES.

Aneurism of the ascending aorta with jerking pulse. *Referred to,* 152, *note.*
Extreme ossific disease of the aortic orifice; remarkable and general musical tone in the superficial arteries; dissection.—*Abstract.* 155.
Ossification and contraction of the mitral valves; complete disappearance of murmur before death; dissection. 157.
Extreme contraction of the mitral valve; absence of murmur; dissection. 159.
Mitral murmur, lasting twelve years without impairment of general health. 164.
Long continuance of loud and rough mitral murmur, with apparently good health. *Abstract.* 166.

Anæmia and chlorosis, with disease of the left auricle, and of left auriculo-ventricular opening; dissection. 167, 517.

Organic disease of the heart, with anæmic and chlorotic symptoms. 167, 515.

Great obstruction of aortic opening from ossific deposits; latency and subsequent sudden development of symptoms; dissection. 169.

Similar case; dissection.—Dr. Graves. 170.

Irregular and excited action of heart, with bellows murmur, without valvular disease; efficacy of an emetic. 177.

Similar case. 178.

Permanent patency of the valves of the pulmonary artery; open foramen ovale; double murmur at the base of the heart, not propagated into the aorta; absence of visible pulsation of the arteries; dissection.—Dr. Gordon. 182.

Dilatation of all the cavities of the heart, of the pulmonary artery, and of the aorta; insufficiency of the auriculo-ventricular valves on both sides; fremitus over the heart, with a musical murmur attending the second sound; replacement of the systolic sound on the left side by a soft murmur: dissection. 184.

Rheumatic fever with endocarditis; signs of confirmed valvular disease; liability during many years to paroxysms of cardiac pains, which are relieved by exercise. 192.

Hypertrophy of heart; universal adherence of pericardium; intense bellows murmur, without valvular disease; dissection.—Dr. Graves. 198.

Disease of the aortic combined with disease of the mitral valves; dissection.—Dr. Law. 199.

Similar double lesion combined with diseased condition of the brain; dissection.—Dr. Law. 199.

Contraction of the mitral and aortic openings; thickening and dilatation of the left ventricle and auricle, the lining membrane of the latter being thickened and opaque: great dilatation of the pulmonary veins; occlusion of the contracted mitral orifice by a coagulum; dissection.—Dr. Adams. 201.

Rounded coagulum, with concentric layers in auricle.—Dr. Adams. *Abstract.* 202.

Permanent patency of the aortic orifice, with contraction and ossification of the mitral valves; dilatation, with hypertrophy of all the cavities of the heart; double bellows murmur at the base of the heart, with a single murmur masking both sounds towards the apex; great enlargement of the right auriculo-ventricular opening; dissection. 204.

Symptoms of valvular disease; occasional dropsy: want of proportion in rate between pulsations of heart and of radial artery; jugular pulsations.—Dr. Adams. 210.

Contraction of mitral opening with permanent rapidity of pulse.—Dr. Adams. 212, *note.*

Similar case.—Dr. Adams. 212, *note.*

Mitral obstruction, with two distinct conditions of heart's action. 212.

Asthma; venous pulsations during paroxysms; arterial coagula; dissection.—Hombert. 215, *note.*

Pulsation in veins of upper extremities; dissection. Dr. Benson. 218.

Jugular pulsation in acute pericarditis. *Abstract.* 220.

Cardiac asthma, with remarkable dulness of left side. *Abstract.* 220.

Sudden development of symptoms of organic disease of the heart; repetition of pseudo-apoplectic attacks, attended by ephemeral hemiplegia and jaundice; dilatation of the left ventricle and auricle; great enlargement of the mitral orifice; dissection.—Dr. Fleming. 222.

Organic disease of the heart; jaundice; pruritus. 224.

Extensive disease of the aortic orifice, with inadequacy of the valves; vast hypertrophy and dilatation of the left ventricle, probably secondary to an attack of endo-pericarditis; aggravated symptoms of angina pectoris, continuing to recur for upwards of ten years: dissection.—Dr. C. Croker King. 234.

Long-existing signs of inadequacy of the aortic valves; persistence of symptoms simulating rheumatic fever; local arterial excitement; cessation of pulsation in the left radial artery; death. 240.

Diseased and inadequate aortic valves; dissection,—Professor Banks. *Abstract.* 244.

Hypertrophy of left ventricle; ossification, shortening, and insufficiency of the aortic valves.—Forget. *Abstract.* 246.

DISEASES OF THE MUSCULAR STRUCTURES OF THE HEART.

Case of Mr. Colles; dilatation of heart with hepatic complication; dissection.—*Abstract.* 278.

Enormous dilatation of right auriole, with some hypertrophy; diastolic pulsation; dissection. *Abstract.* 290.

Violent palpitation of heart; hæmoptysis; enlargement of thyroid gland; death.—Dr. Parry. *Abstract.* 299.

Violent palpitation; pulsating tumour extending high above right clavicle; hæmatemesis; recovery. *Abstract.* 299, *note.*

Palpitation of heart; enlargement of thyroid gland; strong pulsation in carotids; recovery.—Dr. Parry. *Abstract.* 299.

Organic disease of the heart with enlargement of thyroid gland.—Dr. Parry. *Abstracts.* 300.

Palpitation; violent action of carotids; remarkable enlargement and apparent protrusion of eye-balls; tumefaction of thyroid; severe facial neuralgia; death. *Abstract.* 300.

Long-continued excitement of the heart in a male subject; enlargement of the eyeballs and of the thyroid gland; increased action of the vessels of the neck. with murmur in the tumour similar to that of aneurismal varix; ultimate subsidence of the morbid action of the heart, with diminution and hardening of the tumour in the neck. 302.

Cardiac disease; engorgement of veins of neck; enlargement of thyroid gland; gangrene of extremities; dissection.—Sir H. Marsh. *Abstract.* 306.

Hypertrophy of left ventricle; enlargement of thyroid gland; great enlargement of thyroid arteries; dissection.—Professor Smith. *Abstract.* 307.

Great enlargement of thyroid gland with diastolic pulsation; dyspnœa; vast dilatation of the veins of neck; absence of thrill and fremitus. *Abstract.* 307.

Sudden enlargement of the eyes.—Dr. Adams. *Referred to,* 311.

FATTY DEGENERATION OF THE HEART.

Fatty degeneration of both ventricles, with steatomatous and earthy deposits in the aorta; pulse irregular and intermittent; death by apoplexy; dissection.—Dr. Cheyne. 319.

Repeated apoplectic attacks during a long series of years; absence of paralysis; remarkable slowness of pulse; fatty degeneration of both ventricles, especially the right; dissection.—Dr. Adams. 321.

Repeated attacks of syncope; sudden failure of, and total absence of pulse during six weeks; fatty degeneration of the heart; ossification of semilunar valves, aorta, and coronary arteries; dissection.—Dr. Adams. *Abstract.* 323.

Fatty condition of heart; rupture of the left ventricle; free oil in the blood.—Professor Smith. 325.

Fatty condition of the heart; free oil in the blood; dissection.—Professor Smith. 326.

Long-continued palpitation; occasional and sudden faintings; sudden death, with apoplectic symptoms; extensive fatty deposits in the heart; dissection.—Mr. Carmichael. 327.

Anæmic condition; very slow pulse, with valvular murmur; death apparently from syncope; fatty degeneration of the heart, with disease of the aortic orifice; dissection. 328.

Repeated pseudo-apoplectic attacks, not followed by paralysis; slow pulse, with valvular murmur, propagated into the aorta. 329.

Syncopal, merging into apoplectic seizures; symptoms of fatty heart. *Abstract.* 338.

Fatty degeneration of the heart; contraction of the mitral opening; valvular murmur loudest at the apex; feebleness, irregularity, and rapidity of pulse; dissection. 343.

Occasional fainting fits; slight convulsion; single slightly musical bellows murmur attending systole of heart; absence of both sounds. 345.

Fatty heart; enlargement of liver; anæmia. *Abstract.* 348.

Vast abdominal tumour; air in epigastric veins. *Abstract.* 355.

Aneurism of aorta; fatty degeneration and development of air in liver. 355.

Profuse epistaxis; œdema; death; dissection. Air in veins and auricles.—Rérolle de Gex. *Abstract.* 355.

Profuse hemorrhage after operation; emphysema; dissection. Air in veins and right cavities of heart; inflammable gas in muscles.—Rérolle de Gex. *Abstract.* 356.

Profuse epistaxis; extreme debility, with emphysema of subcutaneous cellular membrane of abdomen. Dr. Graves. *Abstract.* 356.

CASES ILLUSTRATIVE OF THE TREATMENT OF THE ORGANIC DISEASES OF THE HEART.

Hypertrophy of heart with valvular disease; severe cardiac asthma superinduced by antiphlogistic treatment, and relieved by tonics. *Abstract.* 367.

Permanent hepatic tumour; alternating periods of tranquillity and excitement of heart; beneficial effects of mercury. *Abstract.* 369.

Hæmoptysis; dyspnœa; cough; disease of aortic and mitral openings; subsequent diminution of heart's action and cessation of abnormal sounds; supervention of paralysis, coma, and death; dissection.—Dr. Law. *Abstract.* 379.

Disease of mitral valves; supervention of pneumonia; occurrence of convulsions and hemiplegia after venesection to four ounces; relief by stimulants; dissection. —Dr. Law. *Abstract.* 379.

THE CONDITION OF THE HEART IN FEVER.

Petechial typhus fever with extreme prostration; continued vigorous action of the heart; failure of the pulse after the eighth day; death on the eighteenth day; complete absence of organic lesion. 400.

Maculated typhus fever, with violent action of the heart; death; no disease discoverable in the heart. 401.

Maculated typhus; absence of the first sound of the heart; extreme slowness of the pulse during convalescence; use of wine in large quantities; recovery. 402.

Maculated typhus; signs of debility of the heart predominating at the left side; absence of impulse; free use of wine; recovery on the seventeenth day. 405.

Maculated typhus, with diminution of the first sound of the heart; use of wine and brandy. 409.

Maculated fever, with severe gastro-catarrhal and nervous symptoms; remarkable modification of the heart's action; use of wine. 410.

Maculated typhus fever; great prostration, with pulmonary and abdominal symptoms; impulse of the heart most distinct with the second sound. 412.

Petechial typhus; the sounds of the heart feeble, but proportionate; loss of impulse continuing for five days, with the progressive restoration of the sounds. 413.

Petechial typhus, with collapse and severe nervous and catarrhal symptoms; great feebleness of the impulse on the twelfth day; vigorous action of the heart for five days before death. 414.

Petechial fever, with bronchitis and diarrhœa; vigorous action of the heart up to the ninth day; preponderance of the first sound on the sixteenth day; use of wine; recovery. 416.

Petechial typhus, with palpitation of the heart and bronchial disease; preponderance of the first sound of the heart; recovery. 417.

Fever of a low character, following on an attack of gastric irritation; absence of the second sound. 418.

Severe maculated fever; delirium; fœtal character of the sounds of the heart; use of wine in large quantities; recovery. 420.

Maculated typhus; loss of impulse on the twelfth, and of both sounds on the thirteenth day; death by syncope on the fifteenth day; extreme softening of the heart. 422.

Adynamic typhus, with severe nervous and pulmonary symptoms; great debility of the heart, with preponderance of the first sound; death on the twelfth day; dissection. 424.

Petechial fever; prostration; varying state of the heart and pulse; death; dissection. 424.

Severe maculated typhus, complicated with intense pulmonary irritation; the sounds of the heart having the fœtal character; employment of wine; death; dissection. 425.

Maculated typhus, with severe nervous symptoms; predominance of the second sound on the sixth day; complete absence of the first sound on the tenth day; death; softened state of the heart; ulceration of the ileum. 427.

Adynamic typhus; vibices; delirium and hæmoptysis; death on the twenty-seventh day; softening of the left ventricle. 430.

Aggravated nervous symptoms in a case of petechial typhus; cessation of the first sound over the left side; extreme softening confined to the left ventricle. 431.

Petechial fever, with intense pulmonary disease; death on the twenty-second day; enlargement and softening of the heart. 433.

Fever; extreme prostration; feeble, irregular action of heart; free use of stimulants; recovery. *Abstract.* 435.

Non-maculated fever, with slight abdominal and pulmonary irritation; absence for several days of the first sound of the heart; second sound distinct with its own impulse; diminished systolic impulse; thrilling pulse; recovery. 435.

Maculated fever; disappearance of an old cardiac murmur, coincident with the signs of softening and weakness of the heart. 438.

Non-maculated fever; relapse, with a cardiac murmur attending the first sound, diminishing in a great degree in the erect position. 440.

Convalescence from measles; murmur with first sound. *Abstract.* 440.

Simple non-maculated fever in an anæmic subject; relapse; development of murmur with the systolic sound. 441.

Simple non-maculated fever; prolongation of the first sound, afterwards passing into a bellows murmur, developed on the seventh day. 442.

Prolonged first sound, passing into cardiac murmur, existing both in the primary fever and in the relapse. 444.

Relapsing fever, with prolongation of the first sound. 444.

Fever, with cardiac murmur in the relapse. 444.

Fever; rough murmur with first sound; organic disease; dissection.—Dr. Lees. *Abstract.* 445.

Cardiac murmur; albuminuria; amenorrhœa; death; organic disease of heart; dissection. 446.

Maculated typhus, with signs of weakness of the heart; musical murmur accompanying the first sound. 448.

Murmur with the first sound of the heart, observed on the twenty-first day, in a case of maculated fever. 450.

Maculated fever; signs of softening of heart; venous congestion of pia mater; unusual quantity of serum in the ventricles of the brain; hepatized appearance of portions of the left ventricle of the heart.—Dr. Hudson. *Abstract.* 457.

Fever; delirium and jactitation; loss of impulse and of first sound; death on the seventeenth day; morbid appearance, as in the preceding case.—Dr. Hudson. *Abstract.* 457.

Fever; debility and softening of heart; muscular tissue of heart infiltrated with blood.—Dr. Hudson. *Abstract.* 457.

Fever; weakness and softened condition of heart.—Dr. Hudson. *Abstract.* 457.

Maculated fever; low delirium; absence of first sound on left side on seventh day; restoration of impulse under the use of wine; convulsions; strong impulse even while sinking; death on tenth day; dissection.—Dr. Hudson. *Abstract.* 458.

Fever; delirium on nineteenth day; impulse of heart strong; pulse weak; death on twenty-third day; cerebral lesions; heart healthy.—Dr. Hudson. *Abstract.* 459.

DISPLACEMENT OF THE HEART.

Diaphragmatic hernia in a man about forty years of age; displacement of heart and mediastinum towards the right side. *Abstract.* 472.

Diaphragmatic hernia in a woman aged seventy-five; displacement of heart to right side; dissection.—Cruveilhier. *Abstract.* 473, *note.*

Diaphragmatic hernia: but little displacement of heart.—Dr. Murphy. *Abstract.* 473.

Chronic bronchitis; sanguineo-serous effusion into pleuræ; swollen state of abdomen; horizontal position of heart.—Dr. Adams. *Abstract.* 479.

RUPTURE OF THE HEART.

Phthisis pulmonalis; pulse at intervals remarkably slow, with bellows murmur attending the first sound; sudden dyspnœa; rupture of left ventricle; death; dissection.—Dr. Bigger. *Abstract.* 483.

Repeated attacks of rheumatism; sudden collapse; death after nineteen hours; rupture of right ventricle; dissection.—Professor Smith. *Abstract.* 485.

Long-existing hypertrophy of the heart, with mitral murmur; acute pleuro-pneumonia, attended with extreme excitement of the heart; sudden occurrence of symptoms of cardiac anguish; death; laceration of the mitral valves and their tendinous cords. 488.

Pneumonia; strong impulse, but confused and irregular action of heart; intense dyspnœa; death; rupture of one of the columnæ carneæ in connexion with the mitral valve,—Corvisart. *Abstract.* 489.

Permanent patency of the aortic valves, with hypertrophy and dilatation of the left ventricle; rupture of the chordæ tendineæ of the anterior portion of the mitral valve.—Dr. Gordon. 489.

Dropsy; considerable impulse of heart; two bellows murmurs of different tone and intensity; hæmatemesis and bloody stools following a wound received upwards of two years before death; hypertrophy of heart, with dilatation of the right ventricle and auricle; rupture of the tricuspid valve.—Dr. Todd. 493.

DERANGED ACTION OF THE HEART.

Gastric derangement; sensation of heat in left arm; subsequent development of bellows murmur; occurrence of pericarditis, followed by signs of hypertrophy. 507.

Production of a murmur during convalescence from maculated typhus; persistence of this phenomenon during the subsequent relapse. 518.

Convalescence from protracted non-maculated fever; development of soft systolic murmur to the left of the nipple; subsequent discovery of loud interscapular murmur; persistence of these signs. *Abstract.* 520.

Measles; distinct systolic murmur, loudest at the apex, probably inorganic. *Abstract.* 521.

Excessive use of green tea; præcordial distress, with tendency to faint; irregular and oppressed respiration; alternation of palpitation with apparently motionless condition of heart; slight fits of apparent asphyxia; relief by opium and stimulants.—Dr. Percival. 533.

Excessive use of green tea; great terror and anxiety; pulse extremely irregular and scarcely discernible; relief by stimulants.—Dr. Harvey. 535.

Abuse of tea; paroxysms of quick and vehement action of the heart; intense præcordial distress and oppression; dread of walking on level surface. 535.

Repeated attacks of rheumatic fever; paroxysms of spasmodic pain in upper sternal region; feeling of soreness, anguish, and agitation about the heart; action of heart regular. 539.

ANEURISM OF THE THORACIC AORTA.

Symptoms of phthisis; arrest of the disease; distinct doubling of second sound. 548.

Physical signs of aneurism of the aorta; great distress in respiration in the horizontal position; recovery. *Abstract.* 555.

Loud and localized aortic murmur; recovery. *Abstract.* 560.

Chlorotic anæmia; singular localization of murmur; recovery under use of protocarbonate of iron. *Abstract.* 560.

Varicose aneurism; vertigo, with temporary loss of vision; orthopnœa; dulness from second to eighth rib; loud bellows murmur with first sound; intense purring tremor; inability to lie on right side; death; communication between pulmonary artery and aorta.—Professor Smith. 570.

Symptoms and signs of chronic phthisis; absence of physical signs of aneurism; tussis clangosa; vast multilocular aneurism of the arch of the aorta. 575.

Aneurism of thoracic aorta; ulcerative perforation of trachea from pressure; absence of stridulous breathing; aphonia.—Professor Smith. *Abstract.* 580.

Aneurism of the transverse portion of the arch of the aorta, extending to the left side; contraction of the side, similar to that following on the absorption of an empyema; displacement of the heart towards the axilla.—Dr. Mayne. 582.

Aneurismal compression of left bronchus; extensive emphysema of trunk and upper extremities; fetid expectoration; sloughing of bronchus and gangrenous condition of lung.—Dr. Greene. *Abstract.* 587.

Aneurism of the arch of the aorta, with perforation of the œsophagus and left bronchial tube; dysphagia and stridor from below, both presenting long intermissions; disappearance of the physical signs of aneurism; death without hemorrhage. 592.

Aneurism of thoracic aorta bursting externally; life prolonged for many days by plugging.—Dr. Osborne. *Abstract.* 596.

Aneurism of thoracic aorta presenting externally; sudden and copious hemorrhages recurring at intervals; death after ten days by exhaustion.—Professor Smith. *Abstract.* 597.

Pulsating tumour in the region of the arch of the aorta; absorption of the ribs and perforation of the integuments; frequent hemorrhages and signs of diminution of the tumour; great improvement of the health under a generous diet.—Dr. Neligan. 598.

Thoracic aneurism; recurrence of copious hemorrhages, attended with convulsions; dissection. *Abstract.* 600.

Emaciation; stridor from below; dulness of clavicles and upper portion of sternum; absence of signs of aneurism; expectoration of milk-white creamy fluid; dissection; numerous cysts surrounding and compressing trachea. *Abstract.* 620.

ANEURISM OF THE ABDOMINAL AORTA.

Deep-seated pain in the back, with neuralgic exacerbations following any change of position; extension of the pain to the intestinal tract; dysphagia; displacement of the liver simulating enlargement; tolerance of enormous doses of opium; dissection.—Dr. Beatty. 627.

Aneurism of the abdominal aorta; double pulsation of the tumour and displacement of the heart. 634.

Aneurism of the abdominal aorta; hemorrhage by successive gushes.—Dr. Lees. 636.

Abdominal aneurism; sudden dulness of left side; rupture into left pleura. *Abstract.* 637.

Bilocular abdominal aneurism; diastolic pulsation and bellows murmur disappearing after a rigor; absence of pulse; effusion of blood into left pleura.—Dr. Law. *Abstract.* 637.

Perforation of stomach without tenderness; and with many of the appearances of hemorrhage from an abdominal aneurism. *Abstract.* 639.

Aneurism of the abdominal aorta; death by perforation of the lung and hæmoptysis. 639.

Symptoms of severe colic, yielding to treatment and short repose, but recurring; sudden death from rupture of an aneurism of the abdominal aorta into the peritoneum. Staff-Surgeon Frazer. 641.

Violent pain in the back, without accompanying morbid signs either local or constitutional; sudden death from rupture of a large abdominal aneurism; carious state of the vertebræ.—Staff-Surgeon Frazer. 641.

Aneurism of the abdominal aorta; formation of pulsating tumours in the left lumbar and iliac regions; sudden death from effusion of blood into the pleura. Dissection. 643.

Aneurism of the abdominal aorta; subperitoneal effusion, forming a pulsating tumour in the left hypochondrium; extensive separation of the peritoneum in the lumbar and iliac regions. 646.

Aneurism of the abdominal aorta; neuralgic pain occurring in paroxysms; effusion of blood beneath the peritoneum covering the liver and stomach; sudden death from effusion into the peritoneum; absence of erosion of the vertebræ. 648.

Aneurism of the abdominal aorta; tumour reaching from the crest of the ilium to the scapula; jaundice; erosion of vertebræ without the peculiar neuralgic pains.—Dr. Hutton. *Abstract.* 649.

Aneurism of the abdominal aorta; diffusion marked by a protracted tremor or rigor.—Dr. Law. *Abstract.* 649.

Vast false aneurism of the abdominal aorta; secondary effusion of blood into the transverse mesocolon; severe neuralgic pains aggravated by exertion; absence of erosion of the vertebræ. 651.

Aneurism of hepatic artery; persistent jaundice; death by sudden rupture into peritoneum; distention of gall-bladder and hepatic ducts with bile. *Abstract.* 654.

Aneurism of abdominal aorta, with erosion of vertebræ; remarkable alleviation of sufferings by violent exercise and pleasurable excitement. *Abstract.* 655.

Non-aneurismal abdominal tumour with diastolic pulsation; peritoneal friction discharge of purulent matter by vomiting and stool; recovery. 660.

INDEX.

ABSCESS, case of hepatic, with opening into pericardial sac, 39; case of acute gangrenous, of lung, with pericarditis, 93; of walls of heart, 128.

Abscesses, pharyngeal, 74 *note.*

Absorption of ribs, case of, 598.

Accumulation, effect of diseases of, in displacing the heart, 469.

Aconite, extract of, in hypertrophy of the heart, 363.

Action, increased, of cervical vessels in pericarditis, 68; met with in but four affections, 69.

—— irregular and excited, of heart, 177; instances of, without valvular lesion, 177; efficacy of emetic in, 177, 178, 179.

—— deranged, of heart, 497.—*See* "Deranged."

Adams, Dr. Robert, crescent-like slit of mitral opening described by, 158, 202; cases of occlusion of mitral orifices by a congulum, 201, 202; observations on disease of mitral valves, referred to, 202, *note;* importance of his contributions to the knowledge of valvular disease, 207; priority of his discoveries, 207, 247; on want of proportion between force of heart and of radial pulse in contraction of mitral orifice, 209: cases of contraction of mitral opening with permanent rapidity of pulse, 212, *note;* on pulsation of jugular veins, 214; on safety-valve function of tricuspid valves, 217, *note*, 496; on form of heart in disease of mitral valves, 232, *note;* on dilatation of auricles, 289; case of sudden enlargement of eye-balls, 311; on atrophy of valves of heart, 315; on weakened state of heart as a cause of apoplexy, 321; case of fatty heart, 321; referred to, 353; on total absence of pulse during six weeks, 324; case of vertical and lateral pressure on the heart, 479.

Adhesion, general, of pericardium, 26; atrophy of heart sometimes coincides with, 28, 111; want of any certain physical sign of, 87; probability of its frequency, 37; effect of, upon heart, 111; case of, 198.

Air, coexistence of, with the usual products of inflammation, a cause of modification of friction signs of pericarditis, 37; temporary existence of, in pericardium, a cause of loudness of heart's sounds, 39; distention of stomach with, modifies all sounds derived from auscultation, 43; posthumous production of, in venous system, in cases of fatty heart, 328; in veins from loss of blood, 355; effect of accumulation of, in displacing the heart, 469, 470.

Air-passages, compression of, from aneurism, 470, 578.

Albuminuria, with fever and cardiac murmur, 446.

Ampullar aneurism of Cruveilhier, 558.

Anæmia, triple combination of acoustic signs in, 153; cases of, with organic disease of the heart, 167, 515, 517.

—— in fatty heart, 348; of brain, a cause of cerebral disease in affections of heart, 377, 378; nervous palpitation in, 510; diagnosis of, 513; case of combination of chlorotic, with organic disease of heart, 515; case of chlorotic, 560.

Anæmic murmurs, existence of, in heart, 157, 548; combination of with organic, 166, 167, 496, *note.*

Andral, M., case of pericarditis simulating angina pectoris, 65; case of small-pox with pericarditis, 95; on pathological statistics of heart in fever, 96; on atheromatous diathesis, 230; on singular augmentation of liver in dilatation of the heart, 284.

Aneurism, simulation of, by disease of aortic valves, 238; double sound in, 288, 554; enlargement of thyroid gland mistaken for, 295; ampullar, of Cruveilhier, 558; mode of death in, 595; advantage of nutritious diet in, 608, *note*, 611; Dr. Mayne's case of varicose, 616; diagnosis between true and false, 623; existence of, without manifest physical signs, 624; three classes of latency of, 624; engaging both abdominal and thoracic cavities, 636.

—— of the abdominal aorta, 626; case of transverse displacement of heart by, 471; Dr. Beatty's case of, 626, 627; displacement of liver in, 629, 630, 633; general history of, 630; modes of death in, 631; atrophy of heart in a case of, 631; frequency of undisturbed pulse and action of heart in, 631; errors in diagnosis in, 632; division of cases of, 633; case of, with double pulsation and displacement of heart, 634; death by convulsions in a case of, 635, 636; case of, with hemorrhage by successive gushes, 636; effect of position on pulsation and murmur of, 637, 659 and *note*; case of, bursting into left pleura, 637; case of bilocular, with effusion into left pleura, 638; case of, with perforation of lung, 639; diagnosis of a case of, from symptoms alone, 640; cases of, with effusion into peritoneum, 641, 648; symptoms attending diffusion of, 642; case of, with formation of pulsating tumours and effusion into pleura, 643; case of, with pulsating tumour, from subperitoneal effusion, 646; probable sources of pain in, 649, 655; case of, with tumour from crest of ilium to scapula, 649; case of diffusion of, attended with rigour, 649; simulation of cancerous tumour by, 650, 651; diagnosis of, obscured by displacement of solid viscera, 653; remarkable alleviation of sufferings in, by violent exercise, 655; diseases which simulate, 656; diagnosis between abdominal tumours and, 657; most frequent seat of perforation in, 658; treatment of, 662; use of chloroform suggested in, 662; recapitulation, 663.

Aneurism of the aorta, extinction of second sound never observed in, 92; Testa's case of dissecting with endo-myocarditis, 127; case of, with three successive ruptures of sac, at intervals of several days, 339.

—— of the thoracic aorta, 553; combination of pericarditis with, 95; jerking pulse in, 152, *note*; sources of diagnosis of, 555; signs of, from percussion, 556; disappearance of signs of, without cure, 557; sounds of, 557; frequent absence of bellows murmur in, 557; behind heart, signs of, 560; double sound and impulse in, 562, 606; effect of position on sound of, 565; regurgitation in, 563, 566; varicose or perforating, 568, 616; modification of signs in varicose, 568; communicating, 568, 616; irregularity of respiration from, 572, 573, 580; occasional latency of, 574; evidences of existence of, 574; occasional absence of pain and of compression of surrounding parts in, 575; case of vast multilocular, with absence of physical signs, 575; characters of pain in, 576; erosion of vertebræ in, 577; pain probably less frequent in true than in false, 578; symptoms of compression from, 578; case of, with contraction of side and displacement of heart, 582; condition of voice in, 584; compression of arteries and veins by, 586; compression of nutrient arteries of lungs by, 587; compression of veins by, 589; compression of œsophagus by, 590; case of, with perforation of œsophagus, and left bronchial tube, 592; relief from use of crutches in, 592, 593; combinations of, 594; consumptive or strumous, 594; with hypertrophy of heart, 595; mode of death in, 595; cases of recurring hemorrhage in, 596, 597, 600; sudden death without rupture in, 601; method of examination in, 603; conditions of heart in, 605; treatment of, 606; venesection in, 609; objections to venesection in, 608, *note*; advantage of occasional leeching in, 610; diet in, 611; probable admissibility of tracheotomy in some cases of, 612; division of attachments of clavicle in, suggested, 612; recapitulation, 612; diagnosis between intrathoracic cancer and, 622; diagnosis between aneurism of innominata and of arch of aorta, 623.

Aneurismal character of impulse in weakened heart, 344, 554, *note*.

—— sounds, characters of, 558.

—— tumour, effects of position on disappearance and reappearance of, 648, 649.

—— varix, murmur resembling that of, in enlarged thyroid gland, 295, 306; case of, 612; purring sensation of, 569, 617.

Angina pectoris, 497; case of pericarditis resembling, 65; case of aggravated symptoms of, during ten years, 234; organic diseases with which it may be combined, 497; symptoms of, 499; nature of, 500; difference between cardiac asthma and, 502; cases so called might often be more properly designated cardiac asthma, 504; desiderata in reference to, 505; treatment of, 505; use of chloroform in, suggested, 506; statistics of, 544; complex or sympathetic functional, of Sir John Forbes, 544.

Antiphlogistic treatment, necessity for caution in the use of, in hypertrophy of the heart, 360; evil of, in hypertrophy with valvular disease, 366; case of severe cardiac asthma induced by, 367; extensive misapplication of, 453, *note*.

Aorta, case of dilatation of, 184; case of remarkably long coagulum in, 215, *note*; case of steatomatous and earthy deposits in, 319; gouty irritation of, mistaken for aneurism, 555; case of constriction of, 560, *note*; communication between the right ventricle and, 569; communication between the pulmonary artery and, 569, 570; pulsating tumour in region of arch of, 598; diagnosis between aneurism of arch of, and of innominata, 623; nervous pulsations of abdominal, 661.

Aortic orifice, extreme ossific disease of, with remarkable musical tone, 155; cases of latent great obstruction of, 169, 170; cases of permanent patency of, 204, 489; case of extensive disease of, 234; permanent patency of, 248; case of disease of, with fatty heart, 328; permanent patency of, mistaken for aneurism, 555.

Aortic valves, division of cases of permanent patency of, 232; case of inadequacy of, 234; case of long existing signs of inadequacy of, 240; variety of duration of first stage of permanent patency of, 248; character of murmur in permanent patency of, 243; case of diseased and inadequate, 244; case of ossification of, 323; lesion of, with fatty heart, 344; case of permanent patency of, 489; permanently patent, mistaken for aneurism, 555, 600.

—— valves, disease of the, 227; signs of, with permanent patency, 152; without permanent patency, 152; combination of, with affection of mitral valves, 199; diminished vital energy in, 230; peculiar character of pneumonia supervening on, 231; physical signs of, 281; increasing and violent pulsations in, 232; form of heart in, 232, *note*; regularity of pulse in, 233; two forms of, 283; excessive use of stimulants in a case of, 236; simulation of aneurism by, 238; case of, with hypertrophy of left ventricle, 246; case of double murmur and single fremitus attending, 524, *note*.

Aortitis, Testa's case of, 127; probable case of gouty, 555.

Apex of heart, direction of, as diagnostic between hypertrophy and nervous excitement of heart, 529.

Aphonia, case of, attending pneumonia, 72; case of, without stridor, in compression of trachea by thoracic aneurism, 580, 584; condition of larynx in aneurismal, 585, *note*.

Apjohn, Dr., on spontaneous combustion, referred to, 356.

Apnœa, in fatty degeneration of the heart, 340.

Apoplexy, Laennec's circumscribed pulmonary, an effect of valvular disease, 194; "nodular" pulmonary, 194; two classes of causes of pulmonary, 203; from fatty degeneration of heart, 338; cases of, 319, 321, 327; treatment of, 328, 338, 339; cause of, 348.

Arcus senilis, in fatty degeneration of the heart, 354.

Arterial obstruction, case of paralysis from, referred to, 324.

Arteries, difference between diseases of and of heart, 146; the "pulse of, unfilled," of Dr. Hope, 152; remarkable and general musical tone in, 155; existence of murmur in, without organic cause, 157; case of polypus occupying, 215, *note*; visible pulsation of, in disease of aortic valves, 228, 330, 555; case of local excitement of, with inadequacy of aortic valves, 240; explanation of sounds in, 266; double sounds in, 267, 268, 554; increased action of, with enlargement of thyroid gland, 294; want of proportion in pulsations of, in affection of heart and thyroid gland, 297, 581; double pulsation in, 298, 564, 566; great enlargement of thyroid, 307; case of ossification of the coronary, 324; white softening of the brain connected by Rostan with disease of the cerebral, 377; connexion between force of pulsations of, and character of second sound, 419; want of consent between heart and, 531; cause of single pulsation of, 565, *note;* compression of, by thoracic aneurism, 586; pulmonary gangrene produced by compression of nutrient of lung, 587.

Arteriosity of veins, 214, *note.*

Artery, case of coagulum occupying the right iliac, and its branches, 131; purring tremor attending dilatation of the pulmonary, 154; case of polypus occupying pulmonary, 215, *note;* case of cessation of pulsation in the radial, 240; displacement of liver by aneurism of hepatic, 633.

Arthritis, cases of apyrexial, rarely attended with pericarditis, 62, 539; case of, with acute pericarditis and pneumonia, 80; case of acute, with pericarditis and double pleuro-pneumonia, 83; Bouillaud's doctrine respecting the heart in, 108; case of, with cardiac complication and bellows murmur, 134; remarkable local irritation simulating, 241.

Ascites, its value as diagnostic between solid tumours and abdominal aneurism, 658, 665.

Asthma, causes of cardiac, 220; probable distention of left auricle in a case of cardiac, 220; with puerile respiration, 341, *note;* case of severe cardiac, induced by antiphlogistic treatment in hypertrophy, 367; difference between cardiac, and angina pectoris, 502; cases of cardiac, sometimes designated angina pectoris, 499, 504.

Atheromatous deposits, occur in both classes of valvular disease, 172, 185; connexion between atheromatous and tuberculous diathesis, 230.

Atrophy, of the heart, 315; of heart in phthisis, 315; of heart, frequently coincides with general adhesion of pericardium, 28, 111; always attends ossification of pericardium, 28; a consequence of formation of false membrane on heart, 111; of stomach, 315; of uterus, 315; of valves of heart, 315; fatty degeneration consequent on, 361, *note;* of lung, a cause of displacement of heart, 475, 476, 478; of vocal muscles from pressure on recurrent nerve, 585, *note;* of heart, in case of abdominal aneurism, 631.

Attrition murmurs of Dr. Hope, 31.

Auricle, M. Forget on relative position of right and left, 161; probable sudden and extraordinary distention of left, in a case of cardiac asthma, 220; case of dilatation of left, 222.

Auricles, dilatation of, 289; dulness and diastolic pulsation in, 290; air in, after profuse hemorrhage, 355.

Auriculo-ventricular valves, their complicated nature and consequent liability to become impaired, 188; action of, 258; rupture of chordæ tendineæ of, 486; probable diagnostics of rupture of, 492.

Auriculo-ventricular opening, case of great enlargement of the right, 204.

Avenbrugger's observation on epigastric tumour produced by eccentric pressure in pericarditis, 58.

B.

Baer, on the effect of position on the chick *in ovo*, 551, *note.*

Bally, M., case of spontaneous combustion, referred to, 356, *note.*

Banks, Professor, case of diseased and inadequate aortic valves, 244; on atrophy of vocal muscles, referred to, 585, *note.*

Bardsley, Sir James, on use of strychnia in cerebral disease, 380.

Bark, its use in gouty and rheumatic pericarditis, 107.

Barlow, Dr., on adherent pericardium, referred to, 111.

Bayle, on rupture of the heart, 485.

Beatty, Dr., case of aneurism of abdominal aorta by, 626, 627; on effect of mental impression in suspending pains of aneurism, 656; on peritoneal friction as attending organic tumours of abdomen, 661.

Bellingham, Dr., on double sound and impulse in aneurism, 562.

Bellows murmur, case of, accompanying first sound in arthritis with cardiac complication, 134; produced by coagulum in left ventricle and aorta in a case of cholera, 140; case of, in hypertrophy of heart, with adherent pericardium, 198; case of double, at base, with single, towards apex of heart, 204; variety of, in maculated fever, 448; at upper portion of left side of chest in phthisis, 546; in acute pleurisy, 547; frequent absence of, in thoracic aneurism, 557, 559.

Bennett, Dr., case of purulent softening of the heart, referred to, 130, *note.*

Benson, Dr., on venous pulsation, 218.

Bertin, on communication between right and left cavities of heart, 181.

Bicuspid valves, action of, in motions of heart, 258.

Bigger, Dr., case of purulent cysts of heart in phthisis, 141; case of rupture of left ventricle, 483.

Billard, on pericarditis in infancy, referred to, 77, *note*.

Bischoff, on spontaneous combustion, referred to, 356 and *note*.

Bizot's measurements of the heart, 272, *note*, and Preface, xxix. *note*.

Bleeding, general, in pericarditis, 100; local in pericarditis, 101; Laennec on atrophy of heart from excessive, 315.

Blisters, use of, in pericarditis, 102.

Blood, influence of arterial, in predisposing to ossific deposits, 181; probable effect of diminished arterial supply of, in producing cerebral symptoms in heart disease, 200, 224, 377, 378; free oil in, 325, 326; emphysema of veins from loss of, 355; transfusion of, in a case of fever, 400; infiltration of muscular tissue of heart with, 457; symptoms of admixture of venous and arterial, 568.

Boerhaave, case of vertical displacement of heart, 471.

Bouillaud, his doctrine in reference to the heart in arthritis, 108; his explanation of the heart's action being perceptible to an increased extent in endocarditis, 119; on cardiac polypi, 130; on purulent cysts of heart, 139; on relative positions of right and left ventricles, 161; on development of muscular fibre in valves of heart, 171 and *note*; on action of columnæ carneæ, 258; on atrophy of heart, 315; on endermic employment of digitalis in hypertrophy of heart, 363; on diaphragmatic hernia, 471; on rheumatic palpitation, 539, 540.

Brain, morbid condition of, in valvular disease, 200; this condition probably dependent on diminished arterial supply, 200, 224, 377, 378; Dr. Law on white softening of, from diseased heart, 377; disorganization of, without paralysis, 377; appears to suffer in typhus from weakened condition of left ventricle, 422; state of, in typhus fever, 455; effects of change of position on pulse in cases of concussion of, 552, *note*.

Bricheteau's case of pneumo-pericarditis, 44, *note*.

Bronchial tubes, compression of, by aneurismal tumour, 572; case of earthy transformation of, 576; case of perforation of left tube, in aneurism of arch of aorta, 592.

Bronchitis, doubling of one of the sounds of the heart in, 527.

Bronchocele, differs from enlargement of thyroid gland with heart disease, 295, 300; Hasse's observations on vascular, 310, *note*.

Broussais, leading practical error of the followers of, 114, 172; too extensive reception of his doctrine even in Britain, 453, *note*; probable explanation of fact relied on by, 466.

Bruit de cuir neuf, 24, 32, 34, 76.

Bruit de soufflet in disease of aortic valves, 228.

Burn, Allan, his work referred to in connexion with pulmonary apoplexy, 194 *note*.

C.

Calomel, its use in pericarditis, 101; Dr. Graves' plan of exhibiting, 101; Dr. Johnson's method of using in diseases of tropical climates, 101.

Cancer, symptoms of intrathoracic, 619; diagnosis of pulsating, 621; simulation of, by abdominal aneurism, 650, 651.

Canton, Mr., on arcus senilis in fatty disease of heart, 354.

Cardiac asthma, causes and physical signs of, 220; remarkable dulness of left side of chest in a case of, 220, 367, 499, 502, 504.

Cardiac disease, connexion between cerebral and, 376.

Cardiac neuralgia, 506; in valvular disease, Dr. Hope's explanation of, 192 and *note*; case of, relieved by exercise, 193; not necessarily connected with valvular disease, 65, 198; treatment of, 508.

Carditis, case of rheumatic, 104; use of wine in rheumatic, 104, 105; local depletion in, 106; mercury in, 106; use of poultices in, 107; necessity for caution in specific treatment in rheumatic, 107; Dr. Latham on use of opium in, 107; Hasse on the rarity of general, in its highest degree, 128; and its frequency in a minor degree, 128; on polypi, as a result of, 130; rarity of, in typhus and typhoid fevers, 520.

Caries of vertebræ, in cases of abdominal aneurism, 641, 645, 648; cases of absence of, in abdominal aneurism, 648, 650; want of diagnostic of, 654, —*See* "Erosion."

Carmichael, Mr., on abscesses posterior to pharynx, referred to, 74, *note*; on treatment of apoplectic symptoms from fatty heart, 328, 339.

Carotid arteries, murmur in, in relapse of fever, 441; obliteration of, in thoracic aneurism, 586.

arswell, Dr., on pulmonary gangrene induced by compression of the nutrient arteries of the lung, 587.

Cases, table of, 667; pericarditis, 667; endocarditis and myocarditis, 668; diseases of the valves, 668: diseases of the muscular structures of the heart, 669; fatty degeneration of the heart, 670; cases illustrative of the treatment of the organic diseases of the heart, 670; the condition of the heart in fever, 671; displacement of the heart, 672; rupture of the heart, 672; deranged action of the heart, 672; aneurism of the thoracic aorta, 673; aneurism of the abdominal aorta, 678.

Cavities of the heart, Laennec, Louis, and Bertin, on communication between the right and left, as predisposing to ossification of valves of right side, 181 and *note;* polypiform concretions in, 202 and *note;* case of dilatation with hypertrophy of all the, 204; diagnosis of valvular disease from state of, 244.

Cavity, double sound produced by a single, 268.

Cerebral arteries, white softening of the brain connected by Rostan with disease of, 377.

Cerebral disease, connexion between cardiac and, 376; use of strychnia in, 380.

Cerebral irritation, coincidence of excitement of heart with, 459.

Cerebral respiration, in fever, 422, 424.

Cerebral symptoms, in heart disease, 200, 224; obliteration of the carotid artery a cause of, in thoracic aneurism, 586.

Cervical vessels, increased action of, in pericarditis, 68; met with in but four affections, 69.

Chevers, Dr., on adherent pericardium, referred to, 111.

Cheyne, Dr., case of fatty heart by, 319; referred to, 353; on imperfect convalescence from fever, referred to, 397.

Child of four months, case of pericarditis in, 77, *note.*

Childhood, pericarditis in, 77, *note.*

Chloroform, its use in angina pectoris suggested, 506; dangerous effects from external use of, in a case of intercostal neuralgia, 506; use of, in abdominal aneurism, suggested, 662.

Chlorosis, doubling of one of the sounds of the heart in, 135; cases of combination of, with organic disease of the heart, 167.

Cholera, coagulum in left ventricle and aorta in a case of, producing bellows murmur, 140, 550.

Chordæ tendineæ, rupture of, 486; in endocarditis, 119; causes of rupture of, 119, 188; Hasse on rupture of, 180: rupture of, a cause of sudden death in valvular disease, 176; use of, 258; mode of distribution of, on the valves, 258; cases of laceration of the, of the mitral valves, 488, 489; probable diagnostics of rupture of, 492; effort of nature at reunion of ruptured, 495.

Churchill, Dr., on pericarditis in infancy, 77, *note.*

Circulation, twofold effect of mitral obstruction on pulmonary, 194; collateral venous, as diagnostic between solid tumours and abdominal aneurism, 658, 665.

Cirrhosis of lung, displacement of heart from, 475, 476.

Clavicle, disarticulation of, suggested in some cases of thoracic aneurism, 612.

Clicking sound in pericarditis, 110.

Climate, greater violence of local inflammation in warm, 128.

Coagula, case of, from arteritis, 131; in left ventricle and aorta in case of cholera, 140.

Coagulum, case of occlusion of mitral orifice by, 201, 202; case of remarkably long, in aorta, 215, *note;* case of long, passing from right ventricle into pulmonary artery, 164.

Colchicum, necessity for caution in the use of, in gouty and rheumatic pericarditis, 107.

Collapsing pulse, 152, *note*, 617 *note.*

Collateral. *See* "Circulation."

Colles, Mr., case of, 278, 368; his character, 280, *note;* on fatty change as consequent on atrophy, 361, *note.*

Collin, first notice of physical signs of pericarditis by, 24; his view of the cause of the friction sound, 24; leather creak sound of, 24, 32, 34, 76.

Columnæ carneæ, action of, 258; use of, 260; comparative immunity of, from softening process, 482; atrophied condition of, 495.

Combustion, spontaneous, suggestion as, to further investigations on the subject of, 356.

Communicating aneurisms, 568; Doctor Mayne's case of, 616.

Compression, symptoms of, from thoracic

aneurism, 578; of air-passages, 578; of arteries and veins, 586; of œsophagus, 590.

Concentric displacement of heart, 474; heart never returns to its natural position after, 477.

Concretions, polypiform, in cavities of heart, 202, *note*.

Concussion of brain, effects of change of position on pulse in, 552, *note*.

Constriction of aorta, case of, 560, *note*.

Consumptive aneurism, 594.

Contraction, case of, of mitral valves with ossification, 157; case of extreme, of mitral valve, without murmur, 159; occurs in both classes of valvular disease, 172; case of, of mitral and aortic openings, 201; case of, of mitral valves, 204.

—— of mitral valves, 207; symptoms, 208; want of proportion between heart, and pulse in, 209; two cases of, with permanent rapidity of pulse, 212, *note*; three groups of cases of, 218; a cause of cardiac asthma, 220; globular form of heart in, 232, *note*, 379; case of, with fatty heart, 348.

—— murmur produced by muscular, 265, 268; of heart, abrupt sharp sound attending, 523; of side, in a case of aneurism of arch of aorta, 582.

Convalescence from fever, retarded or quickened pulse in, 396; Dr. Cheyne on imperfect, 397; case of extreme slowness of pulse in, 402; continuance of prolongation of first sound of heart after, 450; murmur developed during, 450; prolongation of first sound in, 451, 549; practical guidance deducible from effect of change of position on pulse in, 552.

Convulsions, produced by a small bleeding in pneumonia supervening on mitral disease, 379; produced by external use of chloroform, 506; after hemorrhages in thoracic aneurism, 600; death by, in a case of abdominal aneurism, 635, 636.

Cooper, Sir Astley, notice of increased action of cervical vessels in concussion of brain, 69; on effects of change of position on pulse in the same, 552, *note*; on objections to antiphlogistic treatment in aneurism, 609.

Cor bovinum, 232.

Coronary arteries, case of ossification of, 324; obstruction of, in angina pectoris, 502, 503.

Corrigan, Dr., instances of production of extremely indurated false membrane, by acute disease, referred to, 32, *note*; case of coexistence of friction phenomena of pericarditis, with liquid effusion, 36; case of enormous distention of pericardium, referred to, 36, 58; on jerking, collapsing, or regurgitating pulse, 152, *note*; 617, *note*; diagnosis of disease of aortic valves with permanent patency due to, 152, 247; on disease of aortic valves, 228; on cause of death in same, 282; on simulation of aneurism by disease of aortic valves, 288; on murmur in connexion with disease of arteries, referred to, 310; importance of his observations on treatment of combination of hypertrophy of heart with valvular disease, 365; on extraordinary increase of action of heart in this combination, 367; on treatment of inflammatory affections supervening on it, 367; cirrhosis of, 475, 476; on horizontal direction of heart, 480; on ringing sound of heart, 528; on quickness of heart's action, 529, *note*; on direction of apex of heart, 580; on murmur in aneurism, 559; on effects of position on murmur in aneurism, 659, *note*.

Corvisart, his case of epigastric tumour caused by pericardial effusion, 58; case of pericarditis attended with sudden destruction of right eye, 74; case attended with ecchymosis and inflammation of the same, 74; on rupture of the tendinous cords of mitral valves, 488.

Courier in Morgagni, case of the, 78, *note*.

Crampton, Sir Philip, case of pericarditis combined with aneurism of aorta, 95.

Crescent-like slit presented by mitral orifice, 158, 202, 348.

Cretaceous transformation of purulent cysts of the heart, 186, 141.

Crutches, relief from use of, in aneurism, 592, 598.

Cruveilhier, on pericarditis in infancy, referred to, 77, *note*; on purulent cysts of heart, referred to, 186; case of diaphragmatic hernia in a woman of seventy-five, 478, *note*; on deficiency of diaphragm without hernia, 473, *note*; on arrest of development of diaphragm, 474, *note*; on cause of sudden death in rupture of heart, 485; ampullar aneurism of, 553.

Cuir neuf, bruit de, 24, 32, 34, 76.

Cure in disease, retarded by depressing emotions, 38, *note*.

Cutaneous disease, case of disappearance of, followed by acute dry pericarditis, 76.

Cysts, purulent, of heart, 135, *et seq.*, 188, *note.*

D.

Death, modes of, in pericarditis, 20; in dilatation of the heart, 277; in varicose aneurism, 571; in aneurism, 595; in aneurism of abdominal aorta, 631; by convulsions in a case of aneurism of abdominal aorta, 635, 636; sudden, in aneurism of abdominal aorta, from effusion of blood into pleura, 643.

—— rarity of sudden, in disease of the heart, 149; danger of sudden, in valvular disease, 176.

Debility, preponderance of first sound in, in fever, 416, 417.

Degeneration, fatty, of heart, 318; cases of, 319, 321, 323, 325, 326, 327, 328, 343; case of, with total absence of pulse during six weeks, 323; treatment of, 328, 339, 348, 372; two classes of cases of, 332; microscopic appearances of, 333, *note;* two forms of the disease, 334; may occur as a sequel to pericarditis and endocarditis, 335; general diagnosis of, 336; symptoms affecting the nervous system, 338: the respiratory system, 339; and the circulating system, 341; apnœa and sighing respiration in, 340, 341, *note;* division of cases of, into uncomplicated and those combined with disease of valves or aorta, 343; diagnosis of latter combination, 344; its importance, 345; absence of both sounds in, 345; symptoms and signs of, 347; recapitulation, 350; arcus senilis in, 354; value of pedestrian exercise in incipient, 373 and *note;* latent existence of, 375; use of strychnia in, 380; association of, with bronchial diseases, 380.

Delirium, in fever, without cerebritis, 399; cases of, 430, 431, 458, 459; connexion between certain states of heart and in fever, 456.

Deposits, atheromatous and earthy, in both classes of valvular disease, 172, 185; case of steatomatous and earthy, in aorta, 319.

Depression, effect of mental, in retarding cure, 38, *note*, 508; of heart in fever, 402; of heart in fever, followed by excitement, 415; danger of this occurrence, 415.

Deranged action of heart, 497; in fever, 517; in young persons, 531; from derangement of stomach, 532; from use of tobacco, 532, 533; from large doses of sulphate of quina, 538, *note;* from abuse of tea, 538; from hysteria, 537; in rheumatic and gouty diathesis, 538; recapitulation, 542.

Desault, tapping the pericardium practised by, 107.

Dexiocardia, case of, with acute latent pericarditis, 89; case of concentric, from violence, referred to, 475; from rapid absorption of empyema, referred to, 476; two classes of concentric, 478.

Diagnosis, case of, where important, and little value of where difficult, 148, 392, 654, *note;* exclusive, meaning of the term, 187, *note;* of permanent patency of pulmonary valves, Dr. Hope on, 179; difficulty of special, in chronic disease of heart, 187; of double valvular lesion, 205; of organic disease of heart, difficulties of, 509 and *note*, 510, 511; between organic and inorganic murmurs, 549; errors of, in abdominal aneurism, 632; of abdominal aneurism obscured by displacement of solid viscera, 653.

—— of valvular disease, difficulties of, 147; two important points in, 148; in reference to prognosis, 149; difficulty of special, 160, 187, 205, 245; M. Forget's remarks, 161; derived from state of cavities, 244.

Diaphragm, case of protrusion of, in pericarditis, 86; effect of hernia through, in displacing heart, 469, 471; cases of this displacement, 471, 472, 473 and *note;* depression of, in plastic pneumonia, 469; deficiency of, without hernia, 473, *note;* arrest of development of, more common on left than right side, 474, *note.*

Diastolic pulsation, in dilatation of right auricle, 290; in thyroid gland, 308, 310; in semi-fluid abdominal tumours, 656, 657, 660.

Dicrotous pulse, mode of its production, 565, *note.*

Diet, advantage of nutritious, in aneurism, 608, *note*, 611.

Diffuse inflammation, Professor Smith's observations on, 74.

Diffusion of abdominal aneurism, symptoms attending, 642; case of, attended with protracted tremor or rigour, 649.

Digitaline, effects of, on blistered surfaces, 363, *note.*

Digitalis, use of, in pericarditis, 102; endermic employment of, in hypertrophy of heart, 363; best mode of internal exhibition of, in the same, 363.

Dilatation, of cavities of heart, a cause of insufficiency of the valves, 184; of pulmonary artery, rarity of its occurrence, 185; insufficiency of our knowledge in its present state to diagnose dilatation of the pulmonary artery, 186: case of, of left ventricle and auricle and pulmonary veins, 201, 208; *a tergo*, law of, 207; case of, of left ventricle and auricle, 222; of ventricle from valvular inadequacy, 225; case of, of left ventricle, 234; of auricles, 289; of veins of neck, 305, 306, 308.

—— of heart, probably not a merely mechanical result of obstruction, 271; rarity of uncomplicated, 278, 285; met with under two conditions, 278; theoretical diagnosis of, 286; Laennec's statement of differential diagnosis of, 286; murmur in, from insufficiency of valves, 287; recapitulation, 292.

—— and feebleness of heart, with or without valvular disease, 154.

—— of heart with hepatic complication, characters of, 274; varying enlargement of liver in, 274, 275; physical signs of, 275; analogy between condition of liver in, and in diving animals, 275, *note;* mode of death in, 277; beneficial effect of mercurial action in, 278; case of, 278; general view of symptoms of, 288; importance of, 284; suppression of urine in, 285; difficulties of diagnosis under certain circumstances in, 509 and *note*.

—— of heart with hypertrophy, case of, with acute dry pericarditis, 77; case of, with ossification of mitral valves unattended with murmur, 122; case of, of all the cavities, 204; differential diagnosis of, 286; rarity of, in its simple form, 287; theoretical diagnosis of, 288; extension of impulse in, 288; fatty degeneration in, 288; recapitulation, 292; case of, of left ventricle, 489.

—— of side, produced by pericardial effusion, 59; explanation of its comparatively rare occurrence, 59.

Disease, production of extremely indurated false membrane by acute, 82 and *note;* effect of mind on, 88, *note*, 508; valvular, a cause of modification of friction sound in pericarditis, 48; case of disappearance of cutaneous, followed by acute dry pericarditis, 76; difficulty of diagnosis of valvular, 147; rarity of isolation of, in chronic affections of heart, 148; two classes of valvular, 149, 171; causes which concur to produce varied phenomena in cardiac, 156; pathological results of both classes of valvular, 172; cases of combination of organic and functional cardiac, 167, 515, 517; source of sufferings in, 170; different degrees of sufferings in, 602, *note*.

Diseases, of tropical climates, Dr. Johnson's method of using calomel in, 101; of the valves of the heart, 144; of valves at right side of heart, 179; at left, 187; in which careful examination of heart furnishes important indications in treatment, 452; of accumulation, effect of, in displacing the heart, 469; nervous, of heart, 497; which simulate abdominal aneurism, 656.

Displaced heart, acute latent pericarditis affecting, 89; case of intercurrent latent pericarditis in, 92.

Displacement of left lung upwards in pericardial effusion, 59, 60; case of, of liver in pericarditis, 86; of diaphragm and liver, in acute pneumonia, 470; of liver, in aneurism of abdominal aorta, 629, 630, 638; of liver, in aneurism of hepatic artery, 633; of solid viscera, obscures diagnosis of abdominal aneurism, 653.

—— of heart, 468; transverse, 468; eccentric from pressure, 469; vertical, 471, 474; transverse, by aneurism of abdominal aorta, 471; same, from diaphragmatic hernia, 471; concentric, from diminished volume of lung, 474; its bearing on prognosis, 477, 478; does not cause organic disease, 479; does not interfere with the functions of the organ, 474, 479; case of vertical and lateral, 479; in a case of aneurism of arch of aorta, 582; in aneurism of abdominal aorta, 634.

Distention of stomach with air modifies all signs derived from auscultation, 48.

Diving animals, analogy of liver of, to condition of human liver in dilatation of heart with complication, 275, *note*.

Double murmur at base of heart, Dr. Gordon's case of, 182.

—— pulsation, in arteries, 298, 564, 566; in abdominal aneurism, 634, 635.

—— sound, produced by a single cavity, 268; in arteries, 267, 268; in aneurism, 562.

Doubling of one of the sounds of the heart, 182, 526; rarely a cause of modification of friction sound in pericarditis, 45; case of, of second sound in endocarditis, 121; more frequently attends second than first sound, 132, 526; more commonly connected with functional than organic disease, 132, 526, *note;* case of, of second sound in endocarditis, 132; of second sound, in rheumatic endocarditis, 133; of same, while patient retained horizontal posture, in arthritis with cardiac complication, 134; of one of the sounds, in nervous and chlorotic patients, 135; case of, in peripneumonia notha, 135; to be attributed to valvular rather than to muscular action, 135; in nervous disturbance of heart, 526; in endocarditis, 527; in chronic bronchitis, 527; effect of position on, 526, *note*, 527; of second sound, 548.

Dryness of softened heart, 384, 428.

Dynamic condition of heart, dependence of inorganic murmur on, 448; importance of determination of, in many diseases, 452.

Dyspeptic palpitation, 532.

—— pulsation of abdominal aorta, 661.

Dysphagia, a symptom of pericarditis, 69; Testa's case of, 70; probable real character of his cases, 78; sometimes a symptom in thoracic inflammation, 71; case of, attending pleuritis with effusion, 71; two cases of, attending pneumonia, 72; attending aneurism of aorta, 590; case of this complication, 592.

Dyspnœa, in pericarditis, 66; comparison of pericarditis and pleuritis in reference to, 66; with puerile respiration, 341, *note;* absence of, in angina, 502.

E.

Earthy deposits, occur in both classes of valvular disease, 172.

—— transformation of bronchial tubes, case of, 576.

Eccentric pressure, visible signs of, in pericarditis, 58; Avenbrugger's observation of, 58; Louis's case of, 59; Dr. Walshe's explanation of comparative rarity of, 59; occurrence of pericarditis in a heart under influences of, 86, 89, 92.

—— displacement of heart in acute pneumonia, 469.

Effusion, liquid, modifies all the phenomena of pericarditis, but may coexist with friction sounds, 35; Dr. Corrigan's case of, so copious as to simulate empyema, referred to, 58; extrusion of left lung upwards, from extensive, 59; comparison of pericarditis and pleuritis in reference to tolerance of copious, 66; inflammatory, into pericardium without development of friction sound, 79; this occurrence very rare, 80, *note;* treatment of chronic, in pericarditis, 107; tapping pericardium in, 107 and *note;* effect of, into pleura in displacing the heart, 470.

Eggs, effects of position on the hatching of, 551, *note;* reason of their peculiar shape, 551, *note*.

Emetic, efficacy of, in certain cases of excited and irregular action of heart, 177, 178, 179.

Emotions, effect of depressing, in retarding cure in disease, 38, *note*.

Emphysema, of veins from loss of blood, 355; intolerance of antiphlogistic treatment in Laennec's, 381; displacement of heart in Laennec's, 471; case of extensive, produced by aneurismal compression of left bronchus, 587.

Empyema, differences between signs of, derivable from percussion, and those produced by pericardial effusion, 57; Dr. Corrigan's case of pericardial effusion so copious as to simulate, referred to, 58; case of pericarditis, supervening on acute, of the right side, 86; case of extensive, of left pleura with acute latent pericarditis and dexiocardia, 89; cases of chronic, of left pleura with intercurrent latent pericarditis, 92; diminished volume of lung in consequence of, a cause of displaced heart, 475; pulsating, 623.

Endermic employment, of digitalis in hypertrophy of heart, 363; of sedatives generally, further investigations on, suggested, 364.

Endocarditis, 113; variety of circumstances under which it may be observed, 114; formula for its detection, 114; general cannot be distinguished from partial, 115; forms in which the acute disease may be considered, 115; difficulty of diagnosis of chronic, 116; most frequently met with at left side of heart, 116; chief grounds of diagnosis of, 116; difficulty of explaining why the physical signs and more obvious pathological changes of, are confined to the valves, 116; probable cause of frequency of chronic disorganization of the latter, 117; circumstances which render diagnosis of, difficult, 118; frequency of latent occurrence of, 118; probable order of frequency of joint and separate occurrence of pericarditis and endocarditis, 119; symptoms of, 119; Dr. Hope's explanation of the increased extent over which the heart's action is perceptible in, 119; Dr. Law's observation of rupture of chordæ tendineæ in, 119; causes which may produce this accident, 119; occasional absence of valvular murmur in, 120; absence of murmur sometimes probably owing to weakened condition of heart, and sometimes to phlebitic disease, 122; supervention of acute, developing loud murmur, in a case of dilatation and hypertrophy of heart with ossification of mitral valves, previously unattended with murmur, 122; practical conclusions in reference to, 123; case of acute, with doubling of second sound, 132; case of rheumatic, with doubling of second sound, 133; case of, with arthritis and doubling of second sound while the patient retained the horizontal position, 134; inquiry as to whether valvular disease always proceeds from, 171; occurrence of fatty degeneration of the heart as a sequel to, 335; doubling of one of the sounds in, 527.

Endo-pericarditis, history of the discovery of the various murmurs of, 55, *note;* case of hypertrophy and dilatation of the left ventricle probably secondary to, 234.

Enlargement of heart, Dr. Graves' observations on, as a cause of extension of friction sounds in pericarditis, 47, 77, *note.*

Ephemeral conditions of heart in fever, 411.

Epidemics of fever in Ireland, probable unity of, 466.

Epigastric tumours caused by pericardial effusion, 58.

—— tenderness in pericarditis, 65, 66; nature of, still obscure, 66; Doctor Mayne's observations on, 66.

Epistaxis, air in veins after profuse, 355.

Erosion of vertebræ, in cases of abdominal aneurism, 641, 645, 648; want of diagnostic of, 654; probably a painless process, 577, 649, 655; cases of absence of, in abdominal aneurism, 648, 650.

Essentiality of fever, 465.

Excitement, effect of pleasurable, in suspending sufferings in aneurism, 655, 656.

Excitement of heart, case of extreme, with acute pleuro-pneumonia, 488; indicated by ringing sound, 528 and *note;* diagnosis between nervous, and active hypertrophy, 529.

—— of heart in fever, with general debility, unfavourable nature of, 399, 424, 458; single characters of, 401; danger of, supervening on depression, 416, 424; nature of, 419; its danger in advanced stages, 419; case of, 430; feeble action succeeding to, 459, *note;* coincidence of, with cerebral irritation, 459.

—— local, of arteries, 240, 248.

Exercise, value of pedestrian, in incipient fatty disease of heart, 373; remarkable suspension of sufferings in a case of abdominal aneurism by, 655.

Exertion, effects of over, in suddenly developing symptoms of latent disease, 170.

Extension of friction sounds in pericarditis, caused by increase of volume of heart, 47, 77, *note.*

Extrinsic phenomenon of action of heart, 146.

Extrusion of left lung upwards from extensive pericardial effusion, 59; similar displacement in a mixed case, 60; referred to, 73.

Eye, sudden destruction of, attending pericarditis, 74, 75; ecchymosis and inflammation of, 74, 75.

Eye-ball, enlargement of, and of thyroid gland, 294; case of, 302; nature of the enlargement, 311; case of its sudden occurrence, 311.

F.

Faintings, occasional and sudden, in cases of fatty heart, 327, 345.

False membrane, production of extremely indurated, by acute disease, 32 and *note;* on the heart, a cause of atrophy of the organ, 111.

Fatty degeneration of heart, 318; cases of, 319, 321, 323, 325—328, 343; total absence of pulse during six weeks in a case of, 328; treatment of, 328, 339, 348, 372; two classes of cases of, 332; microscopic appearances of, 333, *note;* two forms of, 334; may occur as a sequel to pericarditis and endocarditis, 335; general diagnosis of, 336; symptoms affecting the nervous, 338; respiratory, 339; and circulating systems, 341; apnœa and sighing respiration in, 340, 341, *note;* division into uncomplicated cases, and those combined with disease of valves or aorta, 348; diagnosis of latter combination, 844; importance of this combination, 345; absence of both sounds in, 345, 346; symptoms and signs of, 347; recapitulation, 850; arcus senilis in, 854; in certain cases consequent on atrophy, 861, *note;* value of pedestrian exercise in incipient, 373 and *note;* latent existence of, 875; use of strychnia in, 880; association of, with bronchial diseases, 380.

Febrile diseases, non-inflammatory nature of some local affections which arise during the course of, 451.

Feebleness and dilatation of heart, signs of, with or without valvular disease, 154.

Fever, rarity of occurrence of pericarditis in typhus, 96; Andral on pathological statistics of heart in, 96; the author's researches on use of wine and state of heart in, referred to, 103, *note;* Dr. Hudson on connexion between delirium and certain states of heart in, referred to, 103, *note;* 456 and *note;* first sound of heart more usually suspended in, than in the second, 147; condition of heart in, 382; importance of state of heart in reference to treatment of, 891, 399, 406, 454, 456, 459; fœtal character of heart's action in, 395, 403, 406, 408, 411: epidemic of, at Stockholm, 397; unfavourable nature of excitement of heart with general debility in typhus, 399; cases of this combination, 400, 401, 458; absence of impulse of heart in, 402, 405, 408; diminution of first sound in, 409; purulent secretion from nostrils in, 412, 413, *note;* cases of preponderance of first sound in debility of, 416, 417: absence of second sound in, 418; loss of systolic sound in, 421, 435; case of disappearance of an old cardiac murmur in, 438; development of murmur of heart in, 439: cases of development of murmur in, 440, 441, 442, 444, 446, 448, 450; circumstances generally attending these cases, 441; general remarks on this murmur, 442: murmur in carotids in, 441; enlargement of spleen in relapse of, 442; cases of cardiac murmur from organic disease in, 445, 446; development of bellows murmur in maculated, 448; rarity of this combination, 448; continuation of prolongation of first sound after convalescence from, 450; importance of effects of stimulants on heart in reference to prognosis in, 455; state of brain in typhus, 455; importance of state of heart in reference to use of opium in, 456; nature of murmurs in typhus and typhoid, 460; essentiality of, 465; probable unity of the epidemics of, in Ireland, 466; nervous palpitation in, 510; occurrence of murmur in, 517; case of development of persistent murmur in convalescence from non-maculated, 520; treatment of these cases, 521; use of green tea suggested for procuring sleep in, 537.—*See* "Heart, action of," "Typhus," "Typhoid."

Fibre, Bouillaud on development of muscular, in valves of heart, 171, *note.*

Fistulous opening into pericardial sac, cases of pneumo-pericarditis from, 39, 41.

Flajani, on combination of heart disease with enlargement of thyroid gland, referred to, 299, *note.*

Flatulence, metallic character imparted to phenomena of chest and heart by, 528.

Fleming, Dr., his paper on pharyngeal abscesses, referred to, 74, *note;* case of great enlargement of mitral orifice by, 222; on globular heart, 232, *note.*

Fœtal character of action of heart in fever, 395, 403, 406, 408, 411, 420, 425, 431, 439, 440; importance of, 408, 454.

Fœtus, position of human, *in utero*, 551, *note.*

Foramen ovale, Dr. Gordon's case of open, 182.

Forbes, Sir John, M. D., on diagnosis of pericarditis, 55, *note;* on pulmonary apoplexy, referred to, 194, *note;* on angina pectoris, 544.

Force of the heart, a cause of modification of friction sound in pericarditis, 46.

Forget, M., on adherent pericardium, 111; on purulent cysts of heart, 141; his remarks on the relative situations of the two sides of the heart, 161; statistics of combination of disease of mitral and aortic valves, 206, *note;* dilatation *a tergo* of, 207, 244.

Frazer, Staff-Surgeon, cases of abdominal aneurism, by, 641.

Fremissement, in disease of aortic valves, 229; *cataire*, 563; case of intense, 570.

Fremitus, 523; case of single, with double murmur, attending disease of aortic valves, 524, *note;* Skoda's statement of existence of diastolic, in disease of mitral valves, 524, *note;* regurgitant, 524 and *note;* disappearance of, under pressure, 525.

Friction sounds, Collin's view of cause of, 24; probable indication of, 24; singular localization of friction phenomena of pericarditis, 82 and *note;* conditions under which they are most intense, 33; modified by treatment, 34; and by pressure over the heart, 35; may coexist with liquid effusion, 35, 36; development of, in simple dry pericarditis, 36; in pericarditis with liquid effusion, 36; modified by coexistence of air with usual products of inflammation, 37; metallic character of, derived from distention of stomach with air, 43; modification of, from pleurisy of left lung, 44; by doubling of one of the sounds of the heart, 45; by influence of force and volume of heart, 46; Dr. Graves' observations on extension of, from increase of volume of heart, 47, 77, *note;* not necessarily extended by internal pressure on heart, 48; modified by valvular disease, 48; recapitulation, 52; modification of, in pericarditis, by respiration, 88; case of disappearance of intense, with nearly complete obliteration of pericardial sac, 89; singular duration of, 93.

Friction, peritoneal, diagnostic between organic tumours and abdominal aneurisms, 661.

Frogs, effect of position on hearts of, 551, *note.*

G.

Gairdner, Dr., on adherent pericardium, 112, 113, *note;* his view of certain cases of hæmatemesis, 597, 654 *note.*

Galen on pulsation of jugular veins, 214, *note.*

Ganglia. discovery of microscopic, on surface of heart, 507, *note.*

Gangrene of heart, Testa on, 126; of lung, displacement of heart in consequence of chronic, 475, 476; pulmonary, produced by compression of nutrient arteries of lung, 587.

Gangrenous abscess of lung, case of acute with pericarditis, 93.

Gas, development of in inflammable, in muscle, 356.

Gastric derangement, nervous palpitation in, 510.

Gendrin, M., on production of sound by sudden stretching of membrane, 254; his explanation of second sound of heart, 266; on diagnosis between true and false aneurisms, 624.

Gland, thyroid, enlargement of, and eyeball with heart affection, 294; this combination mistaken for aneurism, 295; thrill in, 295, 809; causes of this affection, 297; difference between this enlargement and that in ordinary goitre, 295, 800, 809; cases of this affection, 802, 806, 807; classification of these cases, 805; nature of their origin, 808; temporary tumefaction of, 295; diastolic pulsation in, 808; Dr. Parry's observations on the functions of, 311; recapitulation, 312.

Globular form of heart in mitral contraction, 232 *note,* 379.

Globus Hystericus, possible source of, 313.

Gluge on the results of myocarditis, referred to, 129: on atheromatous diathesis, referred to, 230.

Goerlitz, Countess, case of, referred to, 356 *note.*

Goitre, differs from enlargement of thyroid gland attending heart affection, 295, 300, 309.

Gordon, Dr., case of permanent patency of valves of pulmonary artery, 182; case of rupture of tendinous cords of mitral valve, 489.

Gout, case of, preceding weakened condition of heart, 319; rarity of cases of fatal metastasis to heart, 375; nervous palpitation in, 510.

Gouty aortitis, probable case of, 555.

—— diathesis, deranged action of heart in, 538.

—— irritation of aorta, mistaken for aneurism, 555.

Graves, Dr., case of pneumo-pericarditis, 39; his observations on influence of increase of volume of heart in causing extension of friction sounds in pericarditis, 47, 77, *note;* case of extrusion of left lung upwards from extensive pericardial effusion, 59; observation of irregularity of pulse in pericarditis before any direct sign of the affection had occurred, 68; on treatment of pericarditis, 101; his plan of administering calomel in pericarditis, 101; case of inflammation of pulmonary valves with softened heart, 122; case of abscess of heart, 128; case of polypus occupying right iliac artery and its branches, 131; on difficulty of special diagnosis in valvular disease, 197; on transient tumefaction of thyroid gland with affection of heart, 295; on difference between the enlargement of the gland in this affection and in ordinary goitre, 300; on emphysema after profuse hemorrhage, 355, 356; on the use of tartar emetic and opium in fever, referred to, 460 and *note;* on effects of position on the rate and action of the heart, 550.

Greene, Dr., on thoracic aneurism, 553, 577; case of aneurismal compression of left bronchus producing extensive emphysema and fetid expectoration, 587; on aneurismal compression of œsophagus, 590.

Guerin, M., on second sound and impulse in aneurism, 562, 567, *note.*

H.

Hæmatemesis, Dr. Gairdner's view of, certain cases of, 597, 654, *note.*

Hæmoptysis, case of, in fever, 480; in abdominal aneurism, 639.

Harvey, case of laceration of heart, 481.

Hasse, on rarity of general carditis in its highest degree, and its frequency in a minor degree, 128; on results of myocarditis, referred to, 129; on rupture of semilunar valves, 130; on rupture of the chordæ tendineæ, 130; on purulent cysts of heart, 141; on nodular pulmonary apoplexy, 194; on vascular bronchocele, 310, *note;* on carditis, 316; admission of dynamic origin of dilatation of intercostals and diaphragm in pleurisy, 317; on fatty degeneration of heart, 318; on local inflammatory action as connected with extension of tumour of aneurism, 610.

Heart, force and volume of, a cause of modification of friction sound in pericarditis, 46; Dr. Graves' observations on influence of increase of volume of, in causing extension of friction sounds in pericarditis, 47, 77, *note;* internal pressure on, does not necessarily cause extension of friction sound in pericarditis, 48; new researches in acoustic signs proper to muscular contraction of, suggested in reference to diagnosis of pericarditis, 51; Bouillaud's doctrine in reference to the, in arthritis, 108; conditions of the, which should excite attention during rheumatic disease, 109; effect of adhesion of pericardium on, 111; Testa's case of ulceration of left ventricle of, 127; abscess of walls of, 128; cases of purulent softening of, referred to, 130, *note;* polypi of, 130; doubling of one of the sounds of, 182, 526; cases of doubling of one of the sounds of, 182, 183, 184. —*See* "Doubling." Intrinsic and extrinsic phenomena of the action of the, 146; difference between the disease of the, and those of the arteries, 146; importance of the condition of the muscular structure of the, in cardiac pathology, 147; rarity of isolation of disease in chronic affections of the, 148; comparative rarity of sudden death in disease of the, 149; causes which concur to produce varied phenomena in disease of the, 156; M. Forget's remarks on relative situations of the two sides of the, 161; cases of combination of anæmia and chlorosis with organic disease of the, 167, 515, 517; absence of symptoms in confirmed affections of, 168; illustrative case, 169; occasional double pulse of, in mitral obstruction, 210; case of sudden development of organic disease of, 222; form of, in valvular diseases, 232, *note;* globular, 232, *note,* 379; diseases of the muscular structures of the, 271; measurements of the, 272, *note;* question of possibility of paralysis of, from primary lesion of nervous centre, 314; microscopic appearance of healthy, 332, *note;* air in, soon after death, 354; air in, after profuse hemorrhage, 355; treatment of organic diseases of the, 357; vital rather than mechanical condition of, to be the great guide in practice, 358; rarity of fatal metastasis of gout to, 375; white softening of brain traced by Dr. Law to disease of, 377; diseases in which a careful examination of, affords important indications in treatment, 452; importance of effects of stimulants on, in reference to prognosis in fever, 455; importance of state of, in angina pectoris, 505; case of infiltration with blood of muscular tissue of, 457; coincidence of excitement of, with cerebral irritation, 459; extreme contraction of, in tetanus, 481, *note;* Dr. Todd on disease of right side of, 493; angina pectoris considered to be spasm of the, 500; discovery of microscopic ganglia on surface of, 507, *note;* difficulty of diagnosis of organic diseases of, 503, and *note,* 510, 511; effects of position on rate and action of, 550; signs of aneurism behind, 560; conditions of, in thoracic aneurism, 605.

Heart, action of, being heard at a great distance, supposed by some to indicate pneumo-pericarditis, 44, *note;* Dr. Hope's explanation of, being perceptible over an increased extent in endocarditis, 119; excited and irregular, depends rather on condition of organ itself, than on that of valves, 177; efficacy of an emetic in certain cases of such action, 177, 178, 179; want of proportion between radial pulse, and in contraction of mitral orifice, 209; same in functional derangement, 529, 530, 532; occasional double, in mitral obstruction, 210; increased, with enlargement of thyroid gland, 294; case of long-continued excited, with same, 302; extraordinary increase of, in some cases of combination of hypertrophy with valvular disease, 367; fœtal character of, in fever, 393, 395, 396, 398, 411, 420, 425, 431; unfavourable nature of excited, with general debility, 399; cases of this combination, 400, 401, 458; depressed, in fever, 402; remarkable modification of, in fever, 410; danger of depressed, followed by excited, 415; varying, in fever, 424; vigorous, up to period of death, in fever, 434; coincidence of excited, with cerebral irritation, 459; case of extremely excited, with acute pleuro-pneumonia, 488; deranged, 497; deranged, in young persons, 531; deranged, in gastric disturbance, 532; from abuse of tea, 533; from hysteria, 537; in rheumatic and gouty diathesis, 538; effects of position on rate and, 550; these effects less observable in hypertrophy, 550; frequency of undisturbed, in abdominal aneurism, 631, 633.

Heart, atrophy of, 315; in phthisis, 315; frequently coincides with general adhesion of the pericardium, 28; always attends ossification of pericardium, 28; a consequence of formation of false membrane on heart, 111; in a case of abdominal aneurism, 631.

—— cavities of the, Laennec, Bertin, and Louis on communication between right and left as predisposing to ossification of valves of right side, 181, and *note;* coagula found in the, 202 and *note;* case of dilatation with hypertrophy of all the, 204; diagnosis of valvular disease from state of the, 244.

Heart, dilatation of, 273.—*See* "Dilatation."

—— depression of.—*See* "Heart, action of."

—— displaced, 468; acute latent pericarditis, in, 89; case of intercurrent latent pericarditis affecting, 89.—*See* "Displacement of heart."

—— excitement of.—*See* "Heart, action of."

—— in fever, Andral on Pathological statistics of the, 96; the author's researches on the state of the, referred to, 103, *note;* Dr. Hudson on connexion between delirium and certain states of the, referred to, 103, *note;* 456, and *note;* state of, 353, 382.—*See* "Fever."

—— fatty degeneration of, in dilatation with hypertrophy, 288.—*See* "Fatty degeneration of heart."

—— gangrene of, Testa on, and ulceration and rupture, 126.

—— hypertrophy of.—*See* "Hypertrophy of heart."

—— impulse of.—*See* "Impulse."

—— inflammation of, and its membranes, 17; general remarks, 17; pericarditis, 19; endocarditis, 113; myocarditis, 125; Hasse's observation of the rarity of general, in its highest degree, and its frequency in a minor degree, 128.

—— motions of the, action of bicuspid and tricuspid valves in the, 258.

—— murmurs in, existence of anæmic, 157; case of double bellows at base, with single towards apex, 204; in valvular insufficiency from dilatation, 287; development of, in fever, 439; dependence of inorganic, on dynamic condition of heart in fever, 443; anæmic murmurs of, 548.—*See* "Murmurs."

—— nervous diseases of, division of, 497; simple neuralgia of the, 506; doubling of one sound in, 526; metallic ringing attending systole in, 527; case of rheumatic neuralgia of heart, 539.

—— purulent cysts of, 135; obscurity of their history, 135; doctrines entertained in reference to them, 136; attributed by Professor Smith to cardiac phlebitis, 137; case of, occurring in both ventricles, 137; Mr. O'Ferrall's cases of, 138, 139; his opinion as to their nature and causes, 139; Bouillaud's view, 139; obscurity of diagnosis of, 140.—*See* "Purulent cysts of the heart." Case of one found behind superior lamina of mitral valve, 188, *note.*

Heart, rupture of, 481; Testa on, 126; case of, 127—*See* "Rupture."

—— softening of.—*See* "Softening."

—— sounds of, loudness of, produced by temporary existence of air in pericardium, 39; case of pneumo-pericarditis with feebleness of, 41; doubling of one of, a cause of modification of friction sound in pericarditis, 45; extinction of second sound, never observed in cases of aortic aneurism, 96; first sound occasionally weakened and almost extinguished in pericarditis, 96; explanation of this phenomenon, 96; cases of double second sound, in endocarditis, 120, 132, 133, 134; great number of possible causes of the, 144; three principal causes of the, 145; reason of the comparative rarity of alteration of second sound, 146; Skoda's views of the, 254; absence of both in fatty heart, 346; modifications of, in fever, 394; impulse with second sound, in fever, 412; effects of pressure on, 561, and *note.*—*See* "Sounds."

—— diseases of the valves of the, 144; difficulties of diagnosis of, 147; difficulty of distinguishing between, of left and right sides, 160, 179, 180; at right side, 179; on communication between right and left cavities, as predisposing to, of right side, 181, and *note;* at left side, 187; reasons why the latter demand our special attention, 188.

—— weakened condition of, 314; circumstances diagnostic of, in pericarditis, 103; sometimes a probable cause of absence of murmur in endocarditis, 122; two kinds of characteristic signs of, in valvular disease, 175; cause of, in syncope, 314; in pericarditis, 315: physical indications of, in fever, 392; sudden, in fever, 458; angina most commonly occurs in, 501.

Hearts of frogs, effect of position on, 551, *note.*

Heberden, Dr., on angina pectoris, 497.

Hellebore, used to produce palpitation, 533.

Hemiplegia, case of ephemeral, in heart disease, 222; case of, produced by a small bleeding in pneumonia supervening on mitral disease, 379.

Hemorrhage, air in veins and heart after profuse, 355; case of recurring, in aneurism of thoracic aorta, 596, 597, 598, 600; by successive gushes in a case of abdominal aneurism, 636.

Hepatic abscess, Dr. Graves' case of, with opening into pericardial sac, 39.

—— artery, displacement of liver by aneurism of, 633; case of, with persistent jaundice, 654.

Hepatic derangement, nervous palpitation in, 510.

Hepatized appearance of portions of left ventricle, case of, 457.

Hernia, diaphragmatic, effects of in displacing the heart, 469, 471; case of, in a man aged forty, 472; in a woman aged seventy-five, 473, *note;* case of, with little displacement of heart, 473.

Heslop, Dr., observation of effect of position on cardiac murmur in fever, 443; cases of murmur in fever noted by, 518.

Histological research, important use of, 498.

Holland, Dr., on diagnosis of aneurisms of innominata, 624.

Hombert, case of venous pulsation, 215, *note.*

Homolle et Quevenne on digitaline, 363, *note.*

Hope, Dr., attrition murmurs of, 31; his explanation of the singular localization of the friction phenomena of pericarditis, 32, *note;* valvular murmur overrated as an indirect sign of pericarditis by, 50; on diagnosis of pericarditis, 55, *note;* history of the discovery of the various murmurs of endo-pericarditis, 55, *note;* on the dyspnœa in pericarditis, 66: on treatment of pericarditis, 99, *note;* explanation of action of heart being perceptible over an increased extent in endocarditis, 119: on jerking pulse, 152, and *note;* on signs of valvular disease, 153; his view of the causes of the first sound of the heart, 158, *note;* on the period when valvular disease becomes chronic, 173: on diagnosis of permanent patency of the pulmonary valves, 179, 183: on rarity of dilatation of pulmonary artery, 185: symptoms assigned by him as indicative of valvular disease, not distinctive, 191: his explanation of the pain attending valvular disease, 192, and *note;* on nodular pulmonary apoplexy, 194, *note;* his explanation of want of proportion between pulsations of heart and of radial artery in mitral obstruction, 211; on atrophy of heart, 315; on fatty degeneration of heart, 318; on diagnosis of fatty disease of heart, 352; on treatment of hypertrophy of heart, 360; on extract of aconite in hypertrophy of heart, 363; on diagnosis of nervous murmurs, 511; on metallic sound, 528, *note;* on aneurism, 553; on character of aneurismal sounds, 558; on signs of aneurism behind heart, 560, 561, and *note;* cases of communication between aneurism of aorta and right ventricle and pulmonary artery, 568; on characters of nervous pulsations of the abdominal aorta, 662.

Horizontal direction of heart, in a case of lateral and vertical pressure, 480; in hypertrophy with dilatation, 480.

Horse, condition of larynx, in a case of "roaring," 585, *note.*

Houston, Dr., on liver in diving animals, 276, *note;* on displacement of heart by tumours in lungs, 471.

Hudson, Dr., on connexion between delirium and certain states of the heart in fever, referred to, 103, *note,* 456; on use of opium in fever, 456, 460; on feeble action succeeding to excitement of heart, 459, *note.*

Hughes, Dr., on occurrence of bellows murmur at upper portion of left side of chest, 546; on anæmic murmurs of heart, 548; on diagnosis between organic and inorganic murmur, 549.

Humboldt, Baron Von, on the effect of position on the heart of frogs, 551, *note.*

Hunter, Mr., on difference between valves of right and left side of heart, 215; tendency of his writings, 458, *note.*

Huss, Professor, on epidemic of fever at Stockholm, 397; on use of stimulants, 398.

Hutton, Dr., case of abdominal aneurism by, 649.

Hydriodate of potash, inutility of, in hypertrophy of heart, 364.

Hydrocyanic acid, use of, in pericarditis, 102.

Hypertrophie dilatatoire of Forget, 294.

Hypertrophy of the heart, diagnosis of, with dry pericarditis, 77; case of, with dilatation and acute dry pericarditis, 77; case of, with dilatation, and with ossification of the mitral valves, unattended with murmur, 122; of the right ventricle a cause of circumscribed pulmonary apoplexy, 194; case of, with adherent pericardium and bellows murmur, 198; with dilatation, of all the cavities, 204; of ventricle from valvular inadequacy, 226; of left ventricle, case of, 234; case of same with disease of aortic valves, 246; with dilatation, 287; extension of impulse in, 288; fatty degeneration in, 288; rarely confined to a single cavity, 289; of the auricles, 289; treatment of, 359; of passive, 360; of active, 360; incurability of confirmed, 360; rarity of uncomplicated occurrence of, 361; evil of repeated bleeding in, 362 and *note;* extract of aconite in, 363; treatment of simple, 363; inutility of hydriodate of potass in, 364; treatment of the combination of, with valvular disease, 365; evil of antiphlogistic treatment in this combination, 366; extraordinary increase of action of heart in some cases of this combination, 367; treatment of inflammatory attacks supervening on, 367; horizontal direction of heart in, with dilatation and permanently patent aortic opening, 480; case of long-existing, with mitral murmur and pleuropneumonia, 488; case of, of left ventricle, and dilatation, 489; angina rare in active, 501; diagnosis between active, and nervous excitement, 529; effects of position on the rate and action of the heart less observable in, 550, 551; with aneurism of the thoracic aorta, 595.

Hysteria nervous palpitation in, 510; deranged action of heart from, 537; great amount of palpitation in, 538; form of palpitation in, consequent on cessation of menses, 538.

Hysteric throbbing of abdominal aorta, 661.

I.

Ileum, case of ulceration of, in fever, 427.

Iliac artery, case of coagulum occupying the right, 131.

Impulse, extension of, in enlargement of the heart, 288; aneurismal character of, in weakened heart, 344, 554, *note;* modifications of, in weakened heart, in fever, 392; jerking of heart in fever, 401, 402, 422, 431; absence of, in fever, 402, 405, 408, 419, 426, 457; with second sound, 412; want of correspondence between return of, and of first sound, in fever, 413; case of loss of, on twelfth day of fever, 422; varying condition of, 429; want of correspondence between, and first sound, 429; case of diminished systolic, with increase of that of second sound in fever, 435; difference between aneurismal and cardiac, 554; "double-jogging," in aneurism, 561; double, in aneurism, 562.

Indurated false membrane, production of, by acute disease, 32 and *note*.

Infancy, pericarditis in, 77, *note*.

Inflammable gas, development of, in muscular structure, 356.

Inflammation, of the heart and its membranes, 17; general remarks, 17; pericarditis, 19; endocarditis, 113; myocarditis, 125; Professor Smith's observation on diffuse, 74; use of wine, in local, 103; greater violence of local, in warmer climates, 128; symptoms diagnostic of, their loss of value in typhus, 459; reactive, 465; abdominal throbbing symptomatic of intestinal, 661, 662, *note*.

Influenza, effects of, in suddenly developing symptoms of latent disease, 170, 171 and *note*.

Innocuousness, even for many years, of valvular disease, and important practical lessons deducible therefrom, 141.

Innominata, diagnosis between aneurism of, and of arch of aorta, 623; Dr. Holland on diagnosis of aneurism of, 624.

Inorganic murmurs, diagnosis of, 512.

Insufficiency, Dr. Walshe on, of pulmonary valves, 183; of the valves, second form of, 183; of the auriculo-ventricular valves, case of, 184; of the aortic valves, case of long-existing signs of, 240.

Insurance, importance of latency of morbus cordis in reference to, 153.

Intestinal irritation, abdominal throbbing symptomatic of, 661, 662, *note*.

Intrinsic phenomena of action of heart, 146.

Iodine, external application of tincture of, in pericarditis, suggested, 102.

Ireland, probable unity of the epidemic fevers of, 466.

Irregularity of pulse, Dr. Graves' observation of, occurring in an early stage of pericarditis, 68; more closely connected with lesion of muscles than of valves of heart, 191.

Isolation of disease, rarity of in chronic affections of the heart, 148.

Itching of the skin, in a case of heart disease, 224.

J.

Jaundice, case of ephemeral, in heart disease, 222; case of, in organic disease of heart, 224; case of persistent, in aneurism of hepatic artery, 654.

Jerking pulse, 152, 617, *note*, 618; Drs. Hope and Corrigan on, 152, *note*; in aneurism of the aorta, 152, *note*.

Johnson, Dr., his method of using calomel in diseases of tropical climates, 101.

Jugular veins, case of pulsations in, in valvular disease, 210; pulsations of, 214; three morbid phenomena observed in, in organic diseases of heart, 217; cases in which pulsation of, should occur, 218; pulsation of, in acute pericarditis, in a case of fatty heart, 220.

K.

Karnwagen, Dr., of Cronstadt, two cases of tapping the pericardium, 108, *note*.

King, Dr. Charles Croker, case of extensive disease of aortic orifice, 234.

King, Mr. T. W., on safety-valve function of right ventricle, 216, *note*, 496; on venous pulsation, 218; on atrophy of valves of heart, 315.

Kirby, Dr., on objections to antiphlogistic treatment in aneurism, 609, *note*.

L.

Laennec, observation of loudness of heart's sounds produced by temporary existence of air in pericardium, 39; on pneumo-pericarditis, 44, *note*; on latency of chronic valvular disease, 162; on disease of valves in cases of communication between right and left cavities of heart, 181; production of a disease corresponding to his circumscribed pulmonary apoplexy, by valvular disease, 194; on action of columnæ carneæ, 258; on differential diagnosis between simple uncomplicated dilatation of heart and the combination with hypertrophy, 286; on atrophy of heart from excessive bleeding, 315; on fatty degeneration of heart, 318, 336; on asthma with puerile respiration, 341, *note*; on altered state of muscles, 382; on softened heart in fever, 383; on cause of rapidity of pulse during convalescence from fever, 396; metallic tinnitus of, 528.

Lancisi, on pulsation of jugular veins, 214, *note*, 217.

Larynx, condition of voice, and stridor in chronic disease of the, 585; condition of, in aneurismal aphonia, 585, *note;* condition of, in a case of roaring in the horse, 585, *note*.

Latency, to be expected in pericarditis when complicated with essential diseases, 94; Dr. Law's experience referred to in connexion with this point, 94, *note;* illustrative case of small-pox with pericarditis by Andral, 95; of chronic valvular disease, 162; its importance in reference to life insurance, 163; of symptoms in confirmed affections of heart, 168; illustrative cases, 169, 170.

Latent occurrence of pericarditis, frequency of, 60; cases of, 86, 89, 92; explanation of, in these cases, 93.

Latham, Dr., on nature of connexion between pericarditis and rheumatic fever, 63, *note;* on use of opium in pericarditis, 107; on treatment as auxiliary to pathology, 359; on incurability of confirmed hypertrophy of heart, 360; caution against repeated bleeding in hypertrophy of heart, 362, *note;* on laceration of abdominal muscles in tetanus, 481, *note;* on angina pectoris, 497, 498 and *note*, 505; on occurrence of bellows murmur at upper portion of left side of chest, 546; on murmur occurring in last periods of life, 550.

Laudanum, excessive use of, in a case of aortic disease, 236.

Law, Dr., pathological observations in reference to value of percussion in complication of pericarditis with pneumonia or pleuro-pneumonia, referred to, 58, *note;* on latency of pericarditis when combined with chronic tubercular disease, 94; on rupture of chordæ tendineæ in endocarditis, 119, 188; cases of combination of disease of aortic and mitral valves, 199; on diseased condition of brain in valvular disease, 200; on diagnosis of double valvular lesion, 205; on globular heart, 232, *note;* on fatty heart, referred to, 358; on connexion between cerebral and cardiac disease, 377, 586; on nux vomica and arnica in fatty degeneration of heart, 380; on characters of pain in aneurismal erosion of vertebræ, 577, 655; on aneurismal dysphagia, 590; on aneurism engaging both abdominal and thoracic cavities, 636; case of abdominal aneurism with effusion of blood into left pleura, 638.

Law of retro-dilatation, 207, 244.

Leather-creak sound of Collin, 16, 32, 34; case of production of, in acute dry pericarditis within a short time before death, 76.

Leeches, use of, in pericarditis, 101, 102.

Lees, Dr., case of pericarditis in a child of four months, 77, *note;* diagnosis of organic cause of cardiac murmur in fever, 445, 447; diagnosis of aneurism, from the effect of position on its murmur and pulsation, 637.

Le Gallois, his opinion on act of vomiting refuted, 478, *note*.

Level surface, dread of walking on, from abuse of tea, 536.

Liebig, Baron, on spontaneous combustion, referred to, 356 and *note*.

Life insurance, importance of latency of morbus cordis in connexion with, 163.

Liquid effusion, modifies all the phenomena of pericarditis, 35; may coexist with friction sounds, 35; case illustrative of this coexistence, 86.

Liver, case of displacement of, in pericarditis, 86; in acute pneumonia, 470; varying enlargement of, in dilatation of heart, 274, 275, 284; of diving animals, analogy between the, and the human liver in dilatation of the heart, 275, *note;* free oil in the, 326; enlargement of, in fatty heart, 348; treatment of enlargement of, with cardiac complication, 368; nervous palpitation in derangement of, 510; displacement of, simulating enlargement in a case of aneurism of abdominal aorta, 629, 630, 633; simulated enlargement of, in a case of aneurism of hepatic artery, 633.

Lobstein on atheromatous diathesis, 230.

Local affections, nature of some non-inflammatory, which arise during course of febrile diseases, 451.

Loudness of heart's sounds caused by temporary existence of air in pericardium, 39.

Louis, pneumo-pericarditis not described by, 44, *note;* on the value of percussion in the complication of pericarditis with pneumonia or pleuro-pneumonia, 58, *note;* case of dilatation of side from pericardial effusion, 59; on the general symptoms of pericarditis, 64; on the dyspnœa attending pericarditis, 66; case of pericarditis supervening during last month of phthisis, 94, *note;* on valvular disease in cases of communication between right and left cavities of heart, 181; observations on softened heart in fever, 383, 407; on delirium without cerebritis, in fever, 399; on non-inflammatory nature of some local affections arising during the course of febrile diseases, 451; referred to, 460, *note.*

Lung, pleurisy of left, a cause of modification of the friction sound in pericarditis, 44; case of extrusion of, upwards, from extensive pericardial effusion, 59; similar displacement in a mixed case, 60; referred to, 73; case of acute gangrenous abscess of, with pericarditis, 93; enlargement of, in *plastic pneumonia*, 469; eccentric displacement of heart from tumefaction of, 469; displacement of heart from diminished volume of, 474; perforation of, in a case of abdominal aneurism, 639.

Lungs, appear to suffer in typhus from weakened condition of right ventricle, 422; displacement of heart in acute diseases of, 469.

Lyons, Dr., cases of murmur in fever noted by, 518; on double sound and impulse in aneurism, 564; on effect of position in modifying the sound of aneurisms, 564; on diagnosis between true and false aneurisms, 624; on aneurism engaging both abdominal and thoracic cavities, 636; diagnosis of rupture of abdominal aneurism into left pleura, 637.

M.

M'Dowel, Dr. B., case of pneumo-pericarditis from perforation of the sac, 41; referred to, 79.

M'Dowel, Dr. E., his cases referred to in connexion with pyogenic condition, 106; polypiform concretion found in left auricle by, 202, *note.*

Manœuvre to ward off cardiac attacks, 331, 339, 379.

Marsh, Sir Henry, case of thyroid tumour, with vast dilatation of veins, 305, 306.

Mayne, Dr., on the friction sound in pericarditis, 25; on first stage of pericarditis, 30, 64; cases of pericarditis referred to, 43, *note*, 50, *note;* on epigastric tenderness in pericarditis, 66; on the dyspnœa of pericarditis, 66; on inflammatory effusion into pericardium, without development of friction sound, 79; on disturbance of heart's action in rheumatic fever, referred to, 539; his case of varicose aneurism, 616.

Measles, case of cardiac murmur in convalescence from, 440; case of distinct systolic murmur in, 521.

Measurements of the heart, 272, *note*, Preface, xiii. *note.*

Meckel, case of displacement of heart, 471.

Medicine, evils attending the separation of, from surgery, 453, *note;* nature of the profession of, 498, *note.*

Membrane, production of extremely indurated false by acute diseases, 32 and *note.*

Membranes of the heart, inflammation of, 17.

Mens medica, the true, described, 359.

Menses, hysterical palpitation consequent on cessation of, 538; increased action of the abdominal aorta preceding the appearance of, 661.

Mercurial action, beneficial effects of, in hepatic complication of dilatation of heart, 278, 368.

Mercury, its use in pericarditis, 101: Dr. Graves' plan of exhibiting, 101; Dr. Johnson's method of using, in the diseases of tropical climates, 101.

Metallic phenomena of pericardium, two classes of, 48.

—— ringing, attendant on systole of heart, 523, 527.

Metaphonia, 614.

Microscopic appearance of normal heart under, 332, *note;* of fatty heart, 333, *note;* use of, in pathological anatomy, 498.

Mind, effect of, on disease, 38, *note*, 508.

Mitral murmur, cessation of, followed by doubling of second sound in a case of acute endocarditis, 133; case of, lasting twelve years without impairment of general health, 164, 166.

—— obstruction, twofold effect of, on pulmonary circulation, 194; effect of, in producing enlargement of right ventricle, 207; two classes of symptoms of, 208; want of proportion between heart and pulse in, 209; three groups of cases of, 213.

—— opening, crescent-like slit presented by, 158, 202, 343; Skoda on constriction of, 197, *note;* cases of occlusion of, by a coagulum, 201, 202; want of proportion between heart and pulse in narrowing of, 209; two cases of contraction of, with permanent rapidity of pulse, 212, *note;* three groups of cases of contraction of, 213; on diagnosis of permanently patent, yet dilated, 216; contraction of, a cause of cardiac asthma; dilated, a possible cause, 220: case of great enlargement of, attended with ephemeral jaundice, 222; jaundice attendant on contraction of, 224; dilatation and hypertrophy of left ventricle from enlargement of, 226 and *note;* regurgitation an important condition in a class of cases of disease of, 226: case of contraction of, with fatty heart, 343.

Mitral valves, ossification of, unattended with murmur, 122; signs of disease of, 151, 195; case of ossification and contraction of, with disappearance of murmur before death, 157; case of extreme contraction of, without murmur, 159; purulent cyst found behind superior lamina of, 188, *note;* shortening of, 189; symptoms of disease of, 190; want of special symptoms of disease of, 191; combinations of disease of, 199; Dr. Adams' observations on disease of, 202, *note;* case of contraction and ossification of, 204; contraction of, 207; symptoms of this lesion, 208; division of cases of disease of, 222; disease of, without contraction, 222; symptoms of disease of, with dilated orifice, 222; globular heart in disease of, 232, *note,* 379: case of laceration of, 488; Skoda's statement of diastolic fremitus attending disease of, 524, *note.*

Montgomery, Professor, on atrophy of the uterus, referred to, 315.

Moore, Dr., reduction of Bizot's measurements of the heart, 273, *note.*

Morgagni, case of the courier in, 73, *note:* on aneurism of abdominal aorta, referred to, 626.

Murmur, may exist in arteries without organic cause, 157; absence of, in case of extreme contraction of mitral valve, 159; character of, in permanent patency of aortic valves, 243; produced by muscular contraction, 265, 268, 269; case of voluntary production of muscular, 270; in dilatation of heart, from insufficiency of valves, 287; in enlarged thyroid, 295, 302, 309; in carotids, in relapse of fever, 441; effects of position on, in aneurism, 637, 659, and *note.*

Murmur, anæmic, existence of, in heart, 157; combination of, with organic, 166, 167; typhoid, 518.

—— bellows, case of, accompanying first sound in arthritis with cardiac complication, 134; case of, in malignant cholera, produced by large coagulum in left ventricle and aorta, 140; development of, in maculated fever, 448: rarity of this combination, 448; occurrence of, at upper portion of left side of chest in phthisis, 546; in acute pleurisy, 547; frequent absence of, in thoracic aneurism, 557.

—— of heart, Dr. Gordon's case of double, at base, 182; temporary suspension of an old, in fever, 438; development of, in fever, 439, 517; cases of, in fever, 440, 441, 442, 444, 446, 448, 450; circumstances generally attending these cases, 441; effect of position on, in fever, 440, 443, 445, 447, 449, 519, 520; with first sound in convalescence from measles, 440; general remarks on, in fever and relapse, 442; cases of, from organic disease, in fever, 445, 446; in convalescence from maculated fever, 518; development and persistence of, in convalescence from non-maculated fever, 520; in measles, 521; treatment of these cases, 521; sudden development of loud musical, in apparently good health, 522: case of double, with single fremitus, attending disease of aortic valves, 524, *note;* occurring in last periods of life, 549.

—— inorganic, dependence of, on dynamic condition of heart, 443.

—— mitral, cessation of, followed by doubling of second sound in a case of acute endocarditis, 133; case of, lasting twelve years without impairment of general health, 164, 166: case of, with long-existing hypertrophy of heart, 488.

—— nervous, diagnosis of, 511; seat of, 512, 513.

—— rasping, compatibility of long duration of, with apparently good health, 166.—*See* "Rasping."

—— to-and fro, 253.

—— valvular, more frequently found at left than at right side of heart, 150; loudness of, not proportioned to extent of disease, 157; case of disappearance of, before death, 157; diminution and cessation of, in progressive disease, 159, 189; conditions capable of inducing this cessation, 189; with fatty degeneration of heart, 328, 329, 348; possibility of absence of, in combination of valvular disease with fatty heart, 343.

Murmur venous, in fever, 444, 446, 449, 450.

Murmurs, attrition, of Dr. Hope, 81; valvular, overrated by Drs. Hope and Watson as an indirect sign of pericarditis, 50; of endo-pericarditis, history of the discovery of the various, 55, *note;* absence of, in cases of considerable ossific deposits on valves, 123; conditions necessary to the production of, 123; nature of, in typhus and typhoid fevers, 460; anæmic, of the heart, 548; diagnosis between organic and inorganic, 549.

Murphy, Dr., case of diaphragmatic hernia, 473.

Muscle, development of inflammable gas in, 356.

Muscles, altered state of voluntary, in typhus fever, 382; cases of, 423, 431; of involuntary, 383; rupture of voluntary, in tetanus, 481; of abdominal in stricture of urethra, 481, *note;* atrophy of vocal, from compression of recurrent nerve, 585, *note.*

Muscular contraction of heart, new researches suggested in acoustic signs proper to, in reference to diagnosis of pericarditis, 51; murmur produced by, 265, 268, 269; case of voluntary production of such murmurs, 270.

—— fibre, Bouillaud on development of in valves of heart, 171 and *note.*

—— power, of heart, deficiency of, 314.

—— prolongation, 525; connected with weakened state of heart, 525.

—— structure of heart, importance of condition of, in cardiac pathology, 147; case of its infiltration with blood, 457.

Musculi papillares, 258, 260; comparative immunity of, from typhoid softening, 432; atrophied condition of, 495.

Musical tone, remarkable and general, in extreme ossific disease of aortic orifice, 155.

Myocarditis, 125; reason of the little opportunity, which exists of studying its pathological anatomy, 125; cases in which it is most likely to be manifested, 125; case of, 125; leading marks of different stages of, 128; generally attacks the left ventricle, 128; arrangement of results of, 129.

N.

Neck, tippet-like swelling of, 308, 589.

Neligan, Dr., case of pulsating tumour by, 598.

Nerve, atrophy of the vocal muscles produced by compression of the recurrent, 585, *note.*

Nerves of Wrisberg, pain in, in cardiac disease, 508.

Nervous diseases of the heart, 497.

—— disturbance of the heart, doubling of one sound in, 135, 526; diagnosis between, and active hypertrophy, 529.

—— murmurs, diagnosis of, 512.

—— pulsations of abdominal aorta, 661; nature of, 662.

Neuralgia, dangerous effects from external use of chloroform in a case of intercostal, 506; simple, of the heart, 506; in arm or shoulder usually a symptom of intra-thoracic disease, 508; treatment of cardiac, 508; rheumatic, of the heart, case of, 539.

Neuralgic pain.—*See* "Pain."

Neurosis of the heart, 497.

Nixon's, Dr., case of constriction of the aorta, 560, *note.*

Nostrils, purulent secretion from, in fever, 412, 413, *note.*

O.

Obliteration of pericardial sac, case of nearly complete, 89; a cause of atrophy of the heart, 111.

Obstruction, case of extreme, of aorta from ossific deposits, with latency of symptoms, 169; case of paralysis from arterial, 324.

Occlusion of mitral orifice by coagulum, 201, 202.

Œsophagus, compression of, by thoracic aneurism, 590; perforation of, in aneurism of arch of aorta, 592.

O'Ferrall, Mr., his cases of purulent cysts of the heart, 138, 139; his view of their nature and causes, 139; on diminution and cessation of murmur in progressive valvular disease, 159; on cessation of regurgitation and murmur in valvular disease, 189.

Oil, free, in blood, 325, 326, 836; in liver and other viscera, 326.

Opacities, occur in both classes of valvular disease, 172.

Opium, Dr. Latham on use of, in pericarditis, 107; excessive use of, in a case of aortic disease, 236; importance of state of heart in reference to use of, 456, 460, 505; efficacy of, in angina pectoris, 499, 500, 505; strong green tea proposed as an antidote to, 587, *note*.

Organic changes, variety of the causes of, 114.

—— murmurs, combination of, with anæmic, 166, 167; diagnosis of, 512.

Organization, process of, retarded by depressing emotions, 88, *note*.

Organs, dependence of suffering in disease on vital rather than on mechanical condition of, 170; former condition of, to be the great guide in practice, 358; Professor Simpson on the absorption of, 361, *note*.

Ormerod, Dr., on fatty degeneration of the heart, 274, *note*, 332, *note;* on microscopic anatomy of the same, 332; on its diagnosis, 336.

Osborne, Dr., case of aneurism by, 596.

Ossification, of pericardium, always attended with atrophy of heart, 28; case of, of mitral valves, unattended with murmur, 122; of mitral valves, cases of, 157, 204; of aortic valves, indications of, 248; case of, of aorta, semilunar valves and coronary arteries, 823.

Ossific deposits, existence of considerable, on valves without production of murmur, 123; case of extreme obstruction of aorta from, with latency of symptoms, 169; influence of arterial blood in predisposing to, 181.

—— disease, signs of extreme, of aortic orifice, 155; case of, 155; remarkable and general musical tone in, 155; case of extreme obstruction of aorta from with latency of symptoms, 169.

P.

Paget, Dr., on fatty heart, referred to, 274, *note*.

Pain, frequently absent in pericarditis, 65; Dr. Hope's explanation of the attending valvular disease, 192; case of cardiac, relieved by exercise, 193; not necessarily connected with valvular disease, 193; neuralgic, in arm or shoulder, usually a symptom of intrathoracic disease, 508; in thoracic aneurism, characters of, 576; probably less frequent in true than in false aneurism of the aorta, 578; two forms of, in abdominal aneurism, 577, 655, 664; diagnosis of aneurism from character of, 640; neuralgic, occurring in paroxysms, in a case of abdominal aneurism, 648; probable sources of, in abdominal aneurism, 649, 655; absence of, in erosion of vertebræ, 649; remarkable suspension of, by mental impression and exertion, in a case of abdominal aneurism, 655.

Pakenham, Mr., diagnosis of aneurism from peculiar nature of the pain, 640.

Palpitation, seizure of apparently nervous, intervening between two attacks of rheumatic carditis, 104; long-continued, in a case of fatty heart, 327; nervous, 508; difficulty of diagnosis between functional and organic, 509 and *note*, 510, 511; nervous classification of cases of, 510; in young persons, 531; from derangement of stomach, 532; from abuse of tea, 583; great amount of, in hysteric excitement of heart, 537, 538; hysterical, consequent on cessation of menses, 538; rheumatic and gouty, 538.

Paralysis, of heart, question of its possibility from primary lesion of nervous centres, 314; absence of, in apoplexy from fatty heart, 321, 329, 338; from arterial obstruction, 324; absence of, in disorganization of brain, 377, 378; case of, produced by small venesection in pneumonia supervening on mitral disease, 379.

Parry, Dr., cases of enlargement of thyroid gland, with heart disease, 298; observations on functions of thyroid gland, 311; on angina pectoris, 499, 502; on Seneca's case, 546.

Patency, permanent, of valves of heart, produced by organic disease, 150; signs of, with disease of aortic valves, 152; occurs in both classes of valvular disease, 172; characteristic double murmur caused by, 176; of pulmonary valves, Dr. Hope on diagnosis of, 179, 183; of valves of pulmonary artery, Dr. Gordon's case of, 182; of aortic orifice, case of, 204; of dilated mitral opening, diagnosis of, 216; of aortic valves, division of cases of, 232; of aortic valves, duration of first stage, 243; character of murmur in, 243; of aortic valves, case of, 489; of aortic valves, mistaken for aneurism, 555.

Pathological anatomy, connexion between, and practical medicine, 358.

Pedestrian exercise, value of, in incipient fatty disease of heart, 373 and *note*.

Percival, Dr., on angina pectoris, referred to, 503; on abuse of green tea, 533; green tea proposed as an antidote to opium by, 537, *note*.

Percussion, signs discoverable by, in pericarditis, 26, 56; differences between those depending on pericardial effusion and those produced by empyema, 57; value of, in complication of pericarditis with pleuro-pneumonia, 58, *note;* signs of thoracic aneurism from, 556.

Perforating aneurisms, 568.

Perforation, of pericardial sac, cases of, 39, 41; of lung, in a case of abdominal aneurism, 639.

Pericarditis, 19; three forms of, 19; mode of death in, 20; classification of cases of, 22; complications of, 22; variety of products of, 32; vital symptoms and history of, 60; three classes of cases of, 60; frequency of its latent occurrence, 60; general symptoms of, 65; case of, simulating angina pectoris, 65; pain in region of heart frequently absent in, 65; nature of epigastric tenderness in, still obscure, 66; Dr. Mayne's observations on this symptom, 66; Louis, Dr. Mayne, and Dr. Hope on dyspnœa of, 66; comparison of pericarditis and pleuritis in reference to dyspnœa and tolerance of copious effusion, 66; conditions of pulse in, 67; Dr. Graves' observation of the early occurrence of irregularity of pulse in, 68; increased action of cervical vessels in, 68; dysphagia a symptom of, 69; Testa's cases of dysphagia attending, 70; probable real character of these cases, 73; cases of, attended with remarkable changes of voice, 72; case of the courier in Morgagni, 73, *note;* Corvisart's case of, accompanied with sudden destruction of right eye, 74; Corvisart's case of, attended with ecchymosis and inflammation of the same, 74; remarks on these cases, 75; case of acute dry, following the disappearance of a cutaneous disease, 76; dry, with hypertrophied heart, diagnosis of, 77; case of acute dry, with hypertrophy and dilatation of the heart, 77; in children, Dr. Churchill on, 77, *note;* Dr. Lees' example of, in a child of four months, 77, *note;* case of acute, with pneumonia and arthritis, 80; case of, with acute arthritis and double pleuro-pneumonia, 83; case of, supervening on acute empyema of right side, with protrusion of diaphragm and displacement of liver, 86; absence of all the usual symptoms in this case, 89; case of acute latent, with empyema and dexiocardia, 89; case of intercurrent latent, in chronic empyema with displaced heart, 92; explanation of latency of, in certain cases, 93; case of, with acute gangrenous abscess of lung, 93; probably generally more or less latent when complicated with essential disease, 94; Louis' case of, supervening during last month of phthisis, 94, *note;* Dr. Law's experience referred to in connexion with latency of complicated pericarditis, 94, and *note;* Andral's illustrative case of small-pox with pericarditis, 95; case of, combined with aneurism of aorta, 95; single sound in advanced periods of this case, 96; weakening of first sound in pericarditis, 96; explanation of this phenomenon, 96; rarity of occurrence of, in typhus fever, 96; circumstances diagnostic of weakened condition of heart in, 103; weakness of heart in, 315; occurrence of fatty degeneration of heart as a sequel to, 335.

Pericarditis, physical signs of, 23; first notice of, by Dr. Collin, 24; *See* "Friction sounds;" Dr. Mayne's researches, 25, 30; phenomena discoverable by touch, 25; by percussion, 26, 56; physical diagnosis, 29; not directly applicable to very first stage, 29; classification of, 31; circumstances which modify, 31; development of tactile vibration, 36; valvular murmur overrated by Drs. Hope and Watson as an indirect sign, 50; new researches in acoustic signs proper to muscular contraction of heart suggested in reference to diagnosis of pericarditis, 51; history of the discovery of the various murmurs of pericarditis, 55, *note;* signs derivable from percussion, 56; means of diagnosis between dulness caused by pericardial effusion and that produced by empyema, 57; value of signs derivable from percussion in complication of pericarditis with pneumonia or pleuro-pneumonia, 58, *note;* visible signs of eccentric pressure, 58; Avenbrugger's observation of epigastric tumour from effusion, 58; Louis' case of dilatation of præcordial region, 59; Dr. Walshe's explanation of the comparatively rare occurrence of dilatation of the side, 59; Dr. Graves' case of extrusion of left lung upwards from extensive effusion in, 59; similar displacement in a mixed case, 60; referred to, 78; change of situation in rasping sound important in diagnosis between pericarditis and valvular disease, 82; clicking sound described by Dr. Walshe, 110; jugular pulsation in acute pericarditis, 220.

—— in rheumatic fever, liability to, 62; proportionate to severity and obstinacy of the fever, 62; sometimes precedes inflammation of joints, 62; Dr. Latham's observations, 63, *note;* rarely appears in the apyrexial cases, 62; conclusions with reference to connexion of rheumatism with pericarditis, 63; treatment of, 108; arrangement of cases of, in which manifest physical signs appear, 110.

—— traumatic, 97; case of, from discharge of small shot from a gun, 97; peculiarity of the signs in this case, 98.

—— treatment of, 98; necessity for caution in continuing antiphlogistic, 98, 816: great advantage derived from physical examination in, 99; Dr. Hope on, 99, *note;* Dr. Wood on, 100, *note;* blood-letting in, 100; local bleeding in, 101; calomel in, 101; Dr. Graves' mode of administering calomel in, 101; repeated use of leeches in, 102; blisters and tincture of iodine in, 102; use of digitalis and hydrocyanic acid in, 102; use of stimulants in, 103; use of wine in, 104, 105, 106; treatment of chronic effusion, 107; tapping the pericardium in, 107; cases of the operation, 107, *note.*

Pericardium, Dr. Graves' case of hepatic abscess opening into, 39; two classes of metallic phenomena of, 43; Dr. Corrigan's case of effusion into, so copious as to simulate empyema, referred to, 58; effusion into, without development of friction sound, 79; this occurrence very rare, 80, *note;* case of nearly complete obliteration of the sac, 89; operation of tapping, 107, and *note.*

—— adhesion of, 26; enumerated among the causes of hypertrophy and dilatation of the heart, 26, 27, 111; atrophy of heart sometimes coincides with, 28, 111; want of any certain physical sign of, 37; probability of frequency of, 37; effects of, on the heart, 111; Drs. Barlow and Chevers, and M. Forget on, 111; Dr. Gairdner on, 112, 113, *note;* case of universal, 198.

—— ossification of, always attended with atrophy of the heart, 28.

Periodicity, interference of reactive inflammations with, in disease, 466.

Peripneumonia notha, doubling of one of the sounds of the heart in a case of, 135.

Peritoneal friction, diagnostic between organic tumours and abdominal aneurism, 661.

Peritoneum, case of extensive separation of, by abdominal aneurism, 646; sudden death from effusion into, in a case of abdominal aneurism, 648; in a case of aneurism of the hepatic artery, 654.

Peritonitis, supervening on hypertrophy of heart with valvular disease, treatment of, 367.

Phlebitic disease, a possible cause of absence of murmur in endocarditis, 122.

Phlebitis, purulent cysts of heart attributed by Professor Smith to cardiac, 137.

Phthisis pulmonalis, Dr. Bigger's case of purulent cysts of heart occurring in, 141; atrophy of heart in, 315; case of remarkably slow pulse in, 483; occurrence of bellows murmur at upper portion of left side of chest in, 546; case of, with aneurism of arch of aorta, 575; combination of, with aneurism, 594.

Physical diagnosis of pericarditis, 29; not directly applicable to very first stage, 29; Dr. Mayne's researches, 80.

—— signs, importance of want of commensurate, in disease, 625.

Piorry, M., on relative positions of right and left ventricles, 161.

Pleura, effect of effusion into, in displacing the heart, 470; aeriform collections in, 470; cases of rupture of abdominal aneurism into, 637, 638, 643.

Pleuritis, modification of friction sound by, 44; comparison of, with pericarditis in reference to dyspnœa and tolerance of copious effusion, 66; case of, attended with dysphagia, 71; murmur at upper portion of left side of chest in, 546.

Pleuro-pneumonia, value of signs derived from percussion in complication of pericarditis with, 58, *note;* case of double, with acute arthritis and pericarditis, 83; case of acute, with hypertrophy and extreme excitement of heart, 488; metallic character imparted by flatulence to phenomena of a case of double, with pericarditis, 528.

Pneumonia, value of signs derived from percussion in complication of pericarditis with, 58, *note;* two cases of, attended with dysphagia, 72; with aphonia, 72; case of, with acute pericarditis and arthritis, 80; supervening on disease of aortic valves, peculiar characters of, 231; supervening on hypertrophy of heart with valvular disease, treatment of, 367; intolerance of venesection in a case of, supervening on disease of mitral valves, 379; depression of diaphragm in plastic, 469; displacement of liver in acute, 470.

Pneumo-pericarditis, case of, 37; Dr. Graves' case of, from fistulous opening into the sac, 39; Dr. M'Dowel's case of, 41; referred to, 79; feebleness of heart sounds in a case of, 43; heart's action being heard at a great distance supposed by some to indicate the existence of, 44, *note;* meagerness of information given by writers on the subject, 44, *note.*

Polemarchus, case of the wife of, alluded to by Testa, 73, *note.*

Polypi, Rokitansky and Bouillaud on carditic, 130; a possible result of carditis, 131; case of, occupying the right iliac artery and its branches, 131; case of, occupying the pulmonary artery and aorta, 215, *note.*

Polypiform concretions, Dr. Adams on, 202, *note.*

Porter, Dr., his cases of internal aneurism, referred to, 590, *note.*

Position, effect of, on doubling of second sound in a case of endocarditis, 134, 135; relative, of the right and left sides of the heart, 161; danger of patients suddenly resuming the erect, after convalescence, 376; effect of, on murmur of heart in fever, 440, 443, 445, 447, 449, 519, 520; rarity of syncope from erect, in typhus with signs of softened heart, 456; effects of, on pulse, 519, 550; on doubling of sounds of heart, 526, *note;* effects of, on the hearts of frogs, 551, *note;* on the chick *in ovo*, 551, *note;* of the human fœtus *in utero*, 551, *note;* effect of, on the pulse, in concussion of the brain, 552, *note;* practical guidance deducible from effect of change of, on the pulse in convalescence, 552; effect of, on the sound of aneurisms, 565; effects of, on pain in abdominal aneurism, 627, 630. 634, 639, 644, 648; on murmur and pulsation in aneurism, 637, 659 and *note;* on disappearance and re-appearance of aneurismal tumour, 648. 649.

Poultices, use of, in rheumatic pericarditis, 107.

Præcordial region, Louis' case of dilatation of, from pericardial effusion, 59.

Pregnancy, pulsations of abdominal aorta occurring in, 661.

Pressure, effects of, on sounds of heart, 561; occurrence of pericarditis in hearts under the influence of eccentric, 86, 89, 92.

—— internal, on heart does not necessarily cause extension of the friction sound in pericarditis, 48.

—— visible signs of eccentric, in pericarditis, 58; Avenbrugger's observation of, 58; Louis' case of, 59; Dr. Walshe's explanation of the comparative rarity of, 59.

Prognosis, value of accuracy in diagnosis of valvular disease in reference to, 149; circumstances which must influence, in valvular disease, 174; in valvular disease, must depend on condition of muscular portion of heart, 176; importance of effects of stimulants on heart in reference to, in fever, 455; bearing of displacement of heart in reference to, 477.

Prolongation of first sound of heart, in relapse of fever, 442; in convalescence, 451, 549; continuation of, in convalescence from fever, 450; in typhus and typhoid fevers, 460; probable cause of, 518; vermicular sensation of, 519, 525; effect of position on, 451, 519, 520; muscular, 525.

Pruritus, case of, in heart disease, 224.

Pulmonary apoplexy, Laennec's circumscribed, a common result of valvular disease, 194; "nodular," 194; Hasse, on, 194; may be produced by hypertrophy of right ventricle, 194; or by distention of pulmonary veins, 203.

—— artery, purring tremor attending dilatation of, 154; case of dilatation of, 184; rarity of this affection, according to Dr. Hope, 185; insufficiency of the present state of our knowledge to diagnose it, 186; case of polypus occupying, 215, *note;* cases of communication between aneurism of the aorta and the, 569, 570.

—— circulation, twofold effect of mitral obstruction on, 194.

—— disease, frequency of combination of, with fatty degeneration of heart, 389.

Pulmonary gangrene, produced by compression of nutrient arteries of lung, 587.

—— valves, Dr. Hope on diagnosis of permanent patency of, 179, 183; Dr. Gordon's case of permanent patency of, 182; Dr. Walshe on insufficiency of, 183.

—— veins, great dilatation of, 201, 203; a probable cause of pulmonary apoplexy, 203.

Pulsating empyema, 623.

—— tumour, case of, 598; diagnosis of cancerous, 621; case of formation of, in aneurism of abdominal aorta, 643; case of same, from subperitoneal effusion, 646.

Pulsation, sources of thoracic, 619; double, in a case of abdominal aneurism, 634; in aneurism, effect of position on, 637, 659; diastolic in semifluid abdominal tumours, 656, 657, 660.

Pulsations, visible, of arteries, 152, 330; in disease of aortic valves, 228, 565; of jugular veins, 210, 214, 331; of jugular veins in acute pericarditis, 220; case of cessation of, in radial artery, 240; diastolic, in dilatation of right auricle, 290; in thyroid tumour, 308, 310; want of proportion in arterial, in affection of heart and thyroid gland, 297; double, in arteries, 298, 564, 566; sympathetic or nervous, of abdominal aorta, 661; nature of these pulsations, 662.

Pulse, conditions of, in pericarditis, 67; Dr. Graves' observation of the early occurrence of irregularity of, in pericarditis, 68; irregularity of, more intimately connected with lesion of the muscles than of the valves of the heart, 191; want of proportion between action of heart and force of radial, in mitral obstruction, 209, 210; similar want of proportion in functional derangement of heart, 529, 530, 532; occasional double, of heart, for single arterial, in mitral obstruction, 210; Dr. Hope's explanation of this phenomenon, 211; in varicose aneurism, 570, 617, *note*, 618; conclusion deducible from existence of a permanently rapid, 218; regularity of, in disease of aortic valves, 233; total absence of perceptible in all parts of body lasting six weeks, 328; tingling, in fatty heart, 342, 343; almost imperceptible during attack of angina pectoris, 503; effects of position on, 519; power of tea in removing irregularity of, 536; effect of position on, in concussion of the brain, 552, *note;* dicrotous, mode of its production, 565, *note;* frequent occurrence of undisturbed, in abdominal aneurism, 681.

—— in fever, diminution of rapidity of, under use of wine, 396, 407, 409; this diminution, and the progressive restoration of the force of the heart attendant on it, only favourable when action of the heart stops short of excitement, 415; progressive retardation of, during convalescence, 396; cause of rapidity of, during convalescence, 396; unfavourable nature of feeble, with vehement action of heart, 399, 400, 401, 424, 458; failure of, after eighth day, 400; case of extreme slowness of, during convalescence, 402; remarkable alternations of, 404; continuation of, at wrist after cessation of impulse of heart, 422: varying condition of, 424; unfavourableness of this condition, 429; case of thrilling, 435; practical guidance deducible from effect of change of position on, in convalescence, 552.

Pulse, jerking, "the pulse of unfilled arteries" of Dr. Hope, collapsing or regurgitant, 152; Dr. Hope on, 152, *note*, 179, 180; Dr. Corrigan on, 152, *note;* in aneurism of aorta, 152, *note;* in varicose aneurism, 617, *note*, 618.

—— slow, the author's memoir on, referred to, 153, 345; remarkably, in cases of fatty degeneration of heart, 321, 328, 329, 342; cause of, in fatty heart, 348; case of extremely, during convalescence from fever, 402; case of remarkably, in phthisis, 483.

—— venous, 214, *note*, 218; in acute pericarditis, 220.

Pulsus formicans, in fatty heart, 342, 343.

Purring tremor, 563; case of intense, 570.

Purulent cysts of the heart, 135; obscurity of their history, 135; doctrines entertained in reference to them, 136; attributed by Professor Smith to cardiac phlebitis, 137; case of, occurring in both ventricles, 137; two cases of, by Mr. O'Ferrall, 138, 139; Mr. O'Ferrall's opinion as to their nature and causes, 139; Bouillaud's view, 139;

obscurity of their diagnosis, 140; general observations on, 140; Dr. Bigger's case of, 141; cretaceous transformation of, 136, 141; Hasse's statement of their more frequent occurrence at left side, and Forget's opposite conclusion, 141; case by Forget, 141; one found behind superior lamina of mitral valve, 188, *note*.

—— secretion from nostrils in fever, 412, 413, *note*.

—— softening of the heart, cases of, referred to, 130, *note*.

Pyogenic condition, Dr. E. M'Dowel's cases, referred to, in connexion with, 106.

Q.

Quain, Richard, M. D., his views of the nature of fatty disease of the heart, 335, 372, *note;* error as to the author's views of the cause of slowness of pulse in cases of fatty heart, 349; on arcus senilis in fatty disease of the heart, 354.

Quina, sulphate of, deranged action of heart from large doses of, 583, *note*.

R.

Radial artery, cessation of pulsation in left, 240.

Ramollissement avec amincissement of Louis, 315.

Rasping sound, change of situation in, important in diagnosis between pericarditis and valvular disease, 82.

Re-animation, apparent, in cases of fatty heart, 327, 328.

Recurrent nerve, atrophy of vocal muscles produced by compression of, 585, *note*.

Regurgitation, Mr. O'Ferrall on cessation of, in valvular disease, 189; in disease of mitral orifice, 226; in disease of aortic valves, 231; in aneurism, 563, 566.

Regurgitant pulse, 152, *note*, 617, *note*.

Relapses, frequent, of fever, 441; development of cardiac murmur in, 440, 441, 444.

Remak, discovery of microscopic ganglia on heart, 507.

Rérolle de Gex, M., on air in veins and heart after profuse hemorrhage, 355.

Respiration, modification of friction sounds in pericarditis by, 83; irregular, in cases of fatty heart, 320, 321, 340, 341, *note;* sighing, in fatty heart, 340, 341, *note;* sighing, in gastric and hepatic derangement, 341, *note;* sighing, in fever, 422, 424; sighing, in angina pectoris, 502, 503; in Seneca's case, 546; cerebral, in fever, 422, 424; inequality of, from pressure of aneurismal tumour, 572, 573, 580.

Respiratory symptoms, three classes of, in fatty heart, 352.

Retention of urine, laceration of abdominal muscles in, 481.

Retro-dilatation, law of, 207, 244; varied period at which this change may become available in diagnosis, 246; not a merely mechanical result of obstruction, 247.

Retzius, Professor, on the reduction of volume of the puerperal uterus, 361 and *note*.

Rheumatic diathesis, deranged action of heart in, 538.

—— disease, frequent immunity of heart from valvular disease in apyrexial, 62, 63, 539.

—— endocarditis, case of, with doubling of second sound, 133.

—— fever, liability to pericarditis in, 62; an essential disease, not necessarily co-existing with arthritis, 62, 63, *note;* conditions of heart which should excite attention during, 109; case of simulation of, 240.

Rheumatic pericarditis, case of, 104: use of wine in, 104, 105; local depletion in, 106; mercury in, 106; use of poultices in, 107; necessity for caution in specific treatment of, 107; Dr. Latham on use of opium in, 107; treatment of, 108.

Rheumatism, conclusions with reference to its connexion with pericarditis, 63; treatment of, supervening on hypertrophy of heart with valvular disease, 367.

Ribs, case of absorption of, 598.

Ringing, metallic, attendant on systole of heart, 523, 527.

"Roaring," condition of larynx of horse in a case of, 585, *note*.

Rokitansky, on carditic polypi, 130; on fatty degeneration of heart, 318, 335; on re-active inflammations, referred to, 465.

Rostan, on connexion between white softening of brain and disease of cerebral arteries, 377.

Rupture, of the heart, 481; Testa on, 126, 127; conditions predisposing to, 482; sudden death by syncope not necessarily caused by, 482; causes of sudden death in, 484; seat of the lesion, 485; of mitral valves, 487; probable diagnostics of, of the cords or valves in one of the auriculo-ventricular openings, 492; of the tricuspid valves, 493; of abdominal aneurism into pleura, diagnosis of, 637.

S.

Sac, pericardial, cases of perforation of, 39, 41; case of nearly complete obliteration of, 89.
Safety-valve function of the right ventricle, 216, *note;* of the tricuspid valves, 217, *note.*
Salter, Dr., case of purulent softening of heart, referred to, 130, *note.*
Schmidt, on morbid development of fat, referred to, 374, *note.*
Schuh, Dr., case of tapping the pericardium, 107, *note.*
Schultz, on pathological development of fat, referred to, 364, *note.*
Secretion, purulent, from nostrils in fever, 412, 413, *note.*
Semilunar valves, action of, 262; cases of ossification of, 323, 324.
Senac, tapping the pericardium suggested by, 107.
Seneca, his account of his own case, 545.
Serres, his theory of the viscera in disease, 275, *note.*
Side, dilatation of, from pericardial effusion, Louis on, 59; Dr. Walshe's explanation of its comparatively rare occurrence, 59.
Sighing respiration, in fatty heart, 340; 341, *note;* in gastric or hepatic derangement 341, *note;* in fever, 422, 424; in angina pectoris, 502, 503; in Seneca's case, 546.
Signs, discoverable by touch in pericarditis, 25; by percussion, 26, 56.
—— physical, of pericarditis, classification and modifying circumstances of, 31; importance of want of commensurate, in disease, 625.
—— visible, of eccentric pressure in pericarditis, 58; Avenbrugger's observation of, 58; Louis' case of, 59; Dr. Walshe's explanation of comparative rarity of, 59.
Simpson, Professor, on absorption of organs, 361, *note.*
Simulation of aneurism by disease of aortic valves, 238.
Situation, change in, of rasping sound, important in diagnosis between pericarditis and valvular disease, 82.
Skin, case of disappearance of disease of, followed by acute dry pericarditis, 76; itching of, in heart disease, 224.
Skoda, on intensification of sounds of pulmonary valves, 197, and *note;* his views of production of sounds of heart, 254; on action of bicuspid and tricuspid valves, 258; of the columnæ carneæ, 258; on use of chordæ tendineæ, 258; on action of semilunar valves, 262; his explanation of the sounds in the ventricles, 263; of the first, 264; of the second, 265; of the sounds in the arteries, 266; remarks on his views, 268; on diastolic fremitus in disease of mitral valves, 524, *note.*
Sleep, power of tea in producing, 537.
Slow pulse, the author's memoir on, referred to, 153, 345; in fatty heart, 321, 328, 329; cause of, 348.
Small-pox, Andral's case of pericarditis with, 95.
Smith, Professor R. W., observations on diffuse inflammation, 74; his instances of abscess of heart alluded to, 128: his view of origin of purulent cysts of heart, 137; his observations on varying enlargement of liver in dilatation of heart, 275, *note;* on atrophy of valves of heart, 315: on free oil in blood, 325, 336; on posthumous turgescence of veins in cases of fatty heart, 328; on air in heart, veins, and solid viscera, 354; on origin of fatty heart, 336, 361, *note;* on depression of diaphragm in plastic pneumonia, 469; on laceration of abdominal muscles on stricture of urethra, 481, *note;* case of rupture of right ventricle, 485; case of varicose aneurism, 570; case of ulcerative perforation of trachea from pressure of aneurism, 580; on condition of larynx of horse in a case of "roaring," 585, *note;* case of aneurism fatal by repeated external losses of blood, 597.
Smyly, Mr., transfusion of blood performed by, in a case of fever, 400.
Softening, exsanguineous, of brain, 377, 378.
—— of heart in fever, 383; affects left in preference to right side, 383, 387, 407; dryness attending, 384, 428; not referrible to inflammation, 384; not the result of putrefactive decomposition, 387; recognisable during life, 387; description of, 388; its capability of retrocession, 389; periodicity of its phenomena, 390; danger of, 422; case of extreme, 422; cases of, 427, 483;

case of long duration of, 430; case of circumscription of, 431.

Softening, purulent, of heart, cases of, referred to, 130, *note*.

Solidists, errors of the, 452, *note*.

Sound, abrupt, sharp, attending contractions of the heart, 523; double, in aneurism, 562.

—— first, of heart, occasionally weakened and almost extinguished in pericarditis, 96; case of bellows murmur accompanying, in arthritis with cardiac complication. 134; more frequently altered than second, 146; more usually suspended in typhus fever 147; case in which it could only have proceeded from ventricular systole, and closing of the tricuspid valve, 158, *note;* Dr. Hope's view of the causes of, 158, *note;* occasional difficulty of distinguishing between, and second, 196; causes of, in the ventricles, 264; case of absence of, in fever, 402; diminution of, in fever, indicates use of stimulants, 404; cases of diminution of, 409, *et seq.;* want of correspondence between return of impulse and of, in fever, 413; cases of preponderance of, in debility in fever, 416, 417, 424; loss of, in fever, 419, 421, 457, 458; absence of, on tenth day of fever, 427; case of cessation of, over left side, 431; absence of, during several days, in fever, 435; development of murmur with, in fever, 442, 444, 446, 448, 450; development of murmur with, in relapse of fever, 440, 441, 444; prolongation of, in fever, 442, 444, 451, 460, 518, 519; continuance of prolongation of, after convalescence from fever, 450; prolongation of, in convalescence in several diseases, 451, 549; vermicular sensation observed in cases of prolongation of, 525.

—— rasping, change of situation in, important in diagnosis between pericarditis and valvular disease, 82.

—— second, of heart, extinction of, never observed in cases of aortic aneurism, 96; case of carditis with doubling of, 120; doubling of, more common than of first, 132; case of doubling of, in acute endocarditis, 132; case of same, in rheumatic endocarditis, 133; case of same, occurring while patient in horizontal position, in arthritis with cardiac complication, 134; reason of the comparatively rare alteration of, 146; occasional difficulty of distinguishing between first and, 196; explanation of the, in the ventricle, 265; Gendrin's explanation of, 266; diminution of, in fever, 395; predominance of, in cases of fever, 404, 427, 430; impulse with, in fever, 412; absence of, in fever, 418; connexion between character of, and force of arterial pulsations, 419; case of increase of, with its own impulse, in fever, 435; doubling of, 548.

Sounds of aneurism, 557; characters of, 558.

—— in the arteries, explanation of the, 266.

—— friction.—*See* "Friction sounds."

—— of heart, loudness of, produced by temporary existence of air in pericardium, 39; case of weakening of, in pneumo-pericarditis, 41; metallic character of, derived from distention of stomach with air, 43; doubling of one of the, a cause of modification of friction sound in pericarditis, 45; doubling of one of the, 132.—*See* "Doubling." Great number of the possible causes of the, 144; three principal causes of, 145; Skoda's views of the production of, 254; variety in the intensity, extent, and character of the, in health, 255; and in disease, 256; effects of varying condition of valves in modifying, 257; explanation of, in ventricles, 263; absence of both, in a case of fatty heart, 346; modification of, in weakened heart in fever, 394; case of loss of both, on thirteenth day of fever, 422; case of loss of both, forty-eight hours before death, 433; effects of pressure on, 561.

Spasm, definition of, 500.

Specific treatment of gouty or rheumatic pericarditis, necessity for caution in, 107.

Spine, erosion of, in consequence of aneurism, 577.

Spleen, enlargement of, in relapse of fever, 442; in thorax, case of, 473.

Spontaneous combustion, suggestion as to further investigation of the subject of, 356.

Stanley, Mr., case of purulent softening of heart, referred to, 130, *note*.

Statistics, of pathological alterations of heart in fever, 96; of the combination of disease of aortic and mitral valves, 206, *note;* of angina pectoris, 544.

Stimulants, use of, in pericarditis, 103; enormous use of, in a case of disease of the aortic valves, 239; tolerance of, in fatty disease of heart, 375; in fever, 465; necessity of caution in withdrawing the ordinary, in treatment of disease, 376; importance of state of heart in reference to use of, in fever, 381, 393, 406, 454, 456, 459; diminution of rapidity of heart's action under use of, 396; use of, in the epidemic at Stockholm, 398; use of, in fever, indicated by diminution of first sound of heart, 404; restoration of force of heart by, only favourable when this stops short of excitement, 415, 424; use of diffusible, in aneurism, 608, *note*.

Stockholm, epidemic of fever at, 397.

Stomach, distention of with air, modifies all signs derived from auscultation, 48; unassisted action of, capable of producing vomiting, 478, *note*; nervous palpitation in derangement of, 510, 532.

Stricture of the urethra, laceration of the abdominal muscles in, 481, *note*.

Stridor, from compression of trachea and bronchial tubes, in aneurism, 572; tracheal, 572; *from below*, 572, 579; tracheo-laryngeal, 572, 580; bronchial, 572, 580; from below, case of, attending aneurism of arch of aorta, 592; without aphonia in aneurism of aorta, its importance in diagnosis, 584; in chronic disease of larynx, 585.

Strumous aneurism, 594.

Strychnia, use of, in fatty heart, 380.

Subsultus tendinum, case of intense, in fever, 481.

Sudden death, comparative rarity of, in disease of heart, 149; danger of, in valvular disease, 176; cases of, in fatty heart, 325, 327; cause of, in fatty heart, 348; by syncope, not necessarily caused by rupture of heart, 482; causes of, in rupture of heart, 484; from effusion into peritoneum, in abdominal aneurism, 648, 654.

Suffering in disease, dependent rather on vital than on mechanical condition of organs, 170; different degrees of, 602, *note*.

Surgery, evils attending the separation of, from medicine, 453, *note*.

Swaine's, Dr., translation of Hasse, referred to for case of purulent softening of heart, 130, *note*.

Symptoms, nature of, 499, *note*; vital, of pericarditis, 60; general, of pericarditis, 65; absence of all the usual, in a case of pericarditis, 86, 89; more prominent, of endocarditis, 119; absence of, in confirmed affections of heart, 168; illustrative cases, 169, 170; sudden development of signs and, in these cases, 170; effects of a general disturbing cause in suddenly developing, 171, *note*; of disease of mitral valves, 190; three classes of respiratory, in fatty heart, 352; want of accordance between constitutional state and, in abdominal aneurism, 625, 640.

Syncope, cause of weakness of heart in, 314; death apparently from, in a case of fatty heart, 328; attacks resembling, in fatty heart, 338; cause of these attacks, 348; case of death by, in fever, 422; from erect position, rarity of, in typhus with signs of softened heart, 456; hysterical, 537.

Systolic sound, loss of, in fever, 419, 421. —*See* "Sound, first, of heart."

T.

Table of cases, 667.—*See* "Cases."

Tapping the pericardium, 107; cases of, 107, *note*.

Tea, deranged action of heart from abuse of, 533; its power of restoring regularity to pulse, 536; dread of walking on level surface, from abuse of, 536; power of green, in producing sleep, 537; its use for this purpose in fever suggested, 537; strong green, proposed as an antidote to opium, 537, *note*.

Testa, notice of his work, 69, *note*; his cases of dysphagia in pericarditis, 70; probable real character of these cases, 73; his allusion to the case of the wife of Polemarchus, 73, *note*; on gangrene, ulceration, and rupture of heart, 126, 127; on pulsation of jugular veins, 214, *note*; on displacement of heart, 471.

Tetanus, rupture of voluntary muscles in, 481; extreme contraction of heart in, 481, *note*.

Thickening, occurs in both classes of valvular disease, 172.

Thomson, Dr., on effects of position on the pulse, 551, *note*.

Thoracic pulsation, sources of, 619.

Thorax, effect of tumours in, in displacing the heart, 469, 471; case of lodgement of spleen in, 473.

Throbbing, abdominal, symptomatic of intestinal inflammation, 661, 662, *note*.

Thurnam, Mr., on varicose aneurism, 568.

Thyroid gland, transient tumefaction of, 295; Dr. Parry on the functions of, 311; connexion between, and state of uterine functions, 311.

—— enlargement of the, and eye-balls, with heart affection, 294; mistaken for aneurism, 295; thrill in, 295; causes of, 297; difference between this enlargement and that in ordinary goitre, 295, 300, 309; cases of, 302, 306, 307; classification of these cases, 305; nature of their origin, 308; diastolic pulsation in, 308; recapitulation, 312.

Tinnitus, metallic, of Laennec, 528.

To-and-fro murmur, 253.

Tobacco, deranged action of heart from use of, 532.

Todd, Dr., his work on gout and rheumatism referred to in connexion with treatment of pericarditis, 100, *note;* on rupture of tricuspid valves, 498; on disease of right side of heart, 493; on natural imperfection of tricuspid valves 496; on effects of pressure on sounds of heart, 561; on atrophy of vocal muscles produced by compression of recurrent nerve, 585, *note.*

Tolerance, of copious effusion, comparison of pericarditis and pleuritis in reference to, 66; of stimulants in fatty disease of heart, 375; of use of diffusible stimuli in certain cases of fever, 455.

Touch, phenomena discoverable by, in pericarditis, 25; development of these phenomena in simple dry pericarditis, 36; in pericarditis with liquid effusion, 37.

Townsend, Dr., on pulmonary apoplexy referred to, 194, *note;* on fatty heart, referred to, 353; on rupture of heart, 485.

Trachea, compression of, by aneurismal tumour, 572, 578; case of ulcerative perforation of, from pressure of aneurism, 579.

Tracheal stridor in aneurism, 572, 579.

Tracheotomy suggested in some cases of thoracic aneurism, 612.

Trance, hysterical, 537.

Transformation, cretaceous, of purulent cysts of heart, 136, 141.

Transfusion of blood, in a case of fever, 400.

Traumatic pericarditis, 97; case of, 97.

Treatment, effects of, in modifying friction sounds of pericarditis, 34; of pericarditis, 98; of gouty or rheumatic pericarditis, necessity for caution in specific, 107; of rheumatic pericarditis, 108; of valvular disease, circumstances which must determine the, 174; in valvular disease, must depend on condition of the muscular portions of the heart, 176; necessity for caution in use of antiphlogistic, in hypertrophy of the heart, 360; evil of antiphlogistic, in hypertrophy with valvular disease, 366.

Tremor, purring, attending dilatation of pulmonary artery, 154.

Tricuspid valves, on safety-valve function of, 216, *note*, 496; action of, in motions of heart, 258; rupture of, 493; natural imperfection of, 496.

Tropical climates, Dr. Johnson's method of using calomel in diseases of, 101.

Tubercular disease, latency of pericarditis when combined with, 94; of the lung, displacement of heart in consequence of chronic, 475, 476.

Tuberculous diathesis, connexion between atheromatous and, 230.

Tubes, bronchial, compression of, by aneurismal tumour, 572; case of earthy transformation of, 576.

Tumour, displacement of heart by abdominal, 471; case of pulsating, 598; diagnosis of pulsating cancerous, 621; case of formation of pulsating, from subperitoneal effusion in abdominal aneurism, 646; effects of position on disappearance and reappearance of an aneurismal, 648, 649.

Tumours, in thorax, effect of, in displacing the heart, 469, 471; case of formation of pulsating, in aneurism of abdominal aorta, 643.

—— organic, peritoneal friction diagnostic between, and abdominal aneurism, 661.

Tumours, semi-fluid, more liable than solid to be mistaken for aneurism, 656; diagnosis between, and aneurism of the abdominal aorta, 657.

Turgescence, posthumous, of veins in cases of fatty heart, 328.

Tussis clangosa, in a case of thoracic aneurism, 575.

Typhoid anæmic murmur, 518.

—— fever, less frequently accompanied with signs of softening of heart than typhus, 435; more frequent occurrence of peculiar murmur in, than in typhus, 518, 526; rarity of carditis in, 520.

—— fevers, nature of murmurs in, 460.

Typhus fever, rarity of occurrence of

pericarditis in, 96; first sound more usually suspended in, than second, 147; state of heart in, 353, 382; altered state of voluntary muscles in, 382; softened heart in, 383.—*See* "Softening." Classification of cases of, 391; importance of state of heart in reference to treatment of, 391, 399, 406, 454, 456, 459; physical indications of weakened heart in, 392; modifications of impulse in, 392; sounds, 394; fœtal character of heart's action in, 395, 403, 406, 408, 411; unfavourable nature of excitement of heart with general debility in, 399; cases of this combination, 400, 401, 458; state of depression of heart in, 402; absence of impulse of heart in, 402, 405, 408; diminution of the first sound in, indicates use of stimulants, 404; cases of this diminution, 409; purulent secretion from nostrils in, 412, 413, *note;* excitement of heart succeeding depression in, 415; danger of this occurrence, 415; cases of preponderance of first sound in stage of debility of, 416, 417; contagiousness of, 421; loss of systolic sound in, 421; development of bellows murmur in, 448; rarity of this combination, 448; case of murmur developed during convalescence from, 450; state of brain in, 455; loss of value of diagnostic symptoms of inflammation in, 459; nature of murmurs in, 460; case of murmur in convalescence from maculated, 518; rarity of the occurrence of carditis in, 520.

U.

Ulceration of the heart, Testa on, 126; his case of, 127.

Urethra, laceration of the abdominal muscles in stricture of, 481, *note.*

Urine, remarkable suppression of, in dilatation of heart with hepatic complication, 285; laceration of the abdominal muscles in retention of, 481, *note.*

Uterine function, connexion between and thyroid gland, 311.

Uterus, on the reduction of volume of the puerperal, 361, and *note;* hysterical palpitation consequent on natural cessation of the functions of, 538.

V.

Valsalva, treatment of, 607.

Valve, purulent cyst found behind superior lamina of mitral, 188.

Valves, aortic, signs of disease of, with permanent patency, 152; without permanent patency, 152; disease of, 227; diminished vital energy in disease of, 230; peculiar character of pneumonia supervening on disease of, 231; physical signs in various stages of disease of, 231; increasing and violent pulsations in disease of, 232; division of cases of permanent patency of, 232; form of heart, in disease of, 232, *note;* regularity of pulse in disease of, 233; case of inadequacy of, 234; excessive use of stimulants in a case of disease of, 236; simulation of aneurism by disease of, 238; case of diseased and inadequate, 244; case of disease of, with hypertrophy of left ventricle, 246; atrophy of, in a case of phthisis, 315; disease of, in cases of fatty heart, 328, 344; case of permanent patency of, 489.

—— auriculo-ventricular, case of insufficiency of, 184; complicated nature of, and their consequent liability to become impaired, 188; action of, 258; probable diagnostics of rupture of, 492.

—— of the heart, physical signs and more obvious pathological changes of endocarditis, confined to, 116; probable cause of frequency of chronic disorganizations of, 117; speculation as to their greater liability to inflammatory disease, 117; diseases of the, 144; effects of organic disease on, 150; permanent patency of, 150; Bouillaud on development of muscular fibres in, 171 and *note;* rupture of, a cause of sudden death, 176; instances of irregular and exalted action of heart without lesion of, 177; diseases of, at right side, 179; difficulty of distinguishing between diseases of, at right and left side, 179, 180, 345; dilatation of the cavities a cause of insufficiency of, 183, 184; diseases of, at left side, 187; reasons why these demand our principal attention, 188; twelve pathological conditions which may induce lesion of, 188; difference between the, of right and left side, 215; effects of varying condition of, in modifying sounds of heart, 257; action of bicuspid and tricuspid in motions of heart, 258; action of columnæ carneæ on, 258; atrophy of the, 315.

—— mitral, ossification of, unattended with murmur, 122; signs of organic disease of, 151, 195; case of ossification and contraction of, 157; case of extreme contraction of, without murmur, 159; ultimate result of disease of, 189; views of Mr. O'Ferrall on cessation of murmur while organic disease of, continues, 189; shortening of, 189; symptoms of disease of, 190; want of special symptoms of disease of, 191; Dr. Hope's explanation of pain attending disease of, 192 and *note;* combinations of disease of, 199; with disease of aortic valves, 199; frequency and statistics of this combination, 206 and *note;* Dr. Adams' observations on disease of, referred to, 202, *note;* case of contraction and ossification of, 204; contraction of, 207; symptoms of contraction of, 208: want of proportion between heart and pulse in contraction of, 209; division of cases of disease of, 222; disease of, without contraction, 222; symptoms of disease of, with dilated orifice, 222; globular form of heart in disease of, 282, *note;* rupture of, 486; cases of rupture of, 488, 489.

Valves, pulmonary, Dr. Hope on diagnosis of permanent patency of, 179; Dr. Gordon's case of permanent patency of, 182; Dr. Walshe on insufficiency of, 183.

—— semilunar, Hasse, on rupture of, 130; action of the, 262; cases of ossification of, 323, 324.

—— tricuspid, on safety-valve function of, 216, *note;* rupture of, 493.

Valvular disease, a cause of modification of friction sound in pericarditis, 48; change of situation in rasping sound important in diagnosis between, and pericarditis, 82; chronic, best considered as an affection *sui generis*, 114; innocuousness of, even for many years, and practical lessons deducible therefrom, 141; difficulties in the diagnosis of, 147, 196; two great practical points to be attended to in the investigation of a case of suspected, 148; two kinds of, 149, 171; Dr. Hope on the signs of, 153; diminution and cessation of murmur in progressive, 159; Mr. O'Ferrall's memoir on this subject, referred to, 159; difficulty of special diagnosis of, 160, 179, 180, 196, 245; three forms and seats of, may be diagnosed, 160; latency of chronic, 162; inquiry as to whether it always proceeds from endocarditis, 171; pathological results of both classes of, 172; circumstances under which a case of, may be treated as one of an inflammatory affection, 172; Dr. Hope's view on this subject, 173; occasional arrest of, 173; final result of chronic, 174; important subjects of consideration in, as determining treatment and prognosis, 174; which must depend on condition of muscular portions of heart, 176; two kinds of signs of weakened condition of heart in, 175; irregular and excited action of heart depends less on, than on state of heart itself, 177; instances of such action without, 177; twelve pathological conditions which may induce, 188; Mr. O'Ferrall on cessation of regurgitation and murmur in, 189; want of distinctive symptoms of, 191; those assigned by Dr. Hope not distinctive, 191; his explanation of pain attending, 192 and *note;* cardiac pains not necessarily connected with, 193; a cause of Laennec's circumscribed pulmonary apoplexy, 194; diagnosis of double, 205; case of, with jugular pulsations, 210; diagnosis of, derived from state of cavities, 244; recapitulation, 248; possibility of occurrence of, without murmur, in fatty heart, 343; treatment of combination of hypertrophy of heart with, 365.

Valvular murmur, overrated by Drs. Hope and Watson as an indirect sign of pericarditis, 50; occasional absence of, in endocarditis, 120, 121; case of this occasional absence, 120; absence of, in cases of considerable ossific deposits on valves, 123; conditions necessary to the production of, 123; more frequently found at left than at right side of heart, 150; loudness of, not proportional to extent of disease, 157; cases of, with fatty degeneration of the heart, 328, 329.

Varicose aneurism, 568.

Varix, aneurismal, purring sensation of, 569; murmur resembling that of, in enlarged thyroid gland, 295, 309; case of, 302.

Veins, great dilatation of pulmonary, 201, 203; a probable cause of pulmonary apoplexy, 203; case of pulsation in jugular, in valvular disease, 210; pulsation of jugular, 214, 331; arteriosity of, 214, *note;* morbid phenomena to be observed in jugular, in organic diseases of heart, 217; three cases in which pulsation of jugular should occur, 218; vast dilatation of, in neck, 305, 306, 308; posthumous turgescence of. in fatty heart, 328; air in, soon after death, 354; air in, after profuse hemorrhage, 355; compression of, by thoracic aneurism, 589.

Venesection, employment of, in pericarditis, 100; evil of repeated, in hypertrophy of heart, 362; question of its justifiability in apoplexy from arterial anæmia, 378; convulsions and hemiplegia produced by a small, in pneumonia supervening on mitral disease, 379; in aneurism of aorta, 609; objection to, in aneurism of aorta, 608, *note*.

Venous blood, symptoms of admixture of, with arterial, 568.

—— murmur, in neck in fever, cases of, 444, 446, 449, 450, 460.

Venous pulse, 214, *note*, 218; in acute pericarditis, 220.

Ventricle, case of coagulum in left, in cholera, 140, 550; on relative positions of right and left, 161; theory of stimulation of, to contraction of, 210; the safety-valve function of the right, 216, *note;* dilatation of left, case of, 222; case of vast hypertrophy and dilatation of the left, 234; case of hypertrophy of the left, with disease of aortic valves, 246; case of hypertrophy of left, with thyroid tumour, 307; rupture of left, from fatty degeneration, 319; case of rupture of left, 325; brain in typhus appears to suffer from weakened condition of left, lungs from that of right, 422; right more softened than left, cases of, 423, 424; cases of softening of left, in fever, 424, 426, 430, 433; of left, alone, 431; case of hepatized appearance of portions of left, 457; rupture of left, case of, 483; of right, case of, 485; hypertrophy and dilatation of left, case of, 489; case of communication between aneurism of aorta and the right, 569.

Ventricles, explanation of the sounds in the, 263; of the first, 264; of the second, 265; cases of fatty degeneration of both, 319, 321; diminished thickness of, in softened heart of fever, 384.

Vermicular sensation in prolongation of first sound, 519, 525; connected with weakened state of heart, 525.

Vertebræ, erosion of, in consequence of aneurism, 577, 641, 645, 648; absence of pain in erosion of, 577, 649; want of diagnostic of erosion of, 577, 654; absence of erosion of, in a case of abdominal aneurism, 648, 649; this process probably painless, 655.

Vessels, increased action of cervical, in pericarditis, 68; met with in but four cases, 69.

Vibices, case of, in fever, 430.

Viscera, free oil in, 826; air in solid, soon after death, 356; displacement of solid, obscures diagnosis of abdominal aneurism, 653.

Visible signs of eccentric pressure in pericarditis, 58; Avenbrugger's observation of, 58; Louis' case of, 59; Dr. Walshe's explanation of their comparatively rare occurrence in pericarditis, 59.

Vital condition of organs, dependence of sufferings in disease on, 170; to be the great guide in practice, 358.

—— symptoms and history of pericarditis, 60.

Vocal muscles, atrophy of, from compression of recurrent nerve, 585, *note*.

Voice, loss of, attending pneumonia, 72; remarkable changes of, attending pericarditis, 72; condition of, in aneurism of thoracic aorta, 584; in chronic disease of larynx, 585.

Volume of the heart, a cause of modification of friction sound in pericarditis, 46; Dr. Graves' observations on the influence of increase of, in causing extension of friction sounds, 47, 77, *note*.

Vomiting, may be accomplished by unaided action of stomach, 473, *note*.

W.

Walshe, Dr., his explanation of the comparatively rare occurrence of dilatation of the side in pericarditis, 59; clicking sound in pericarditis described by, 110; on atrophy of heart in consequence of formation of false membrane on its surface, 111; on insufficiency of pulmonary valves, 183; on nodular pulmonary apoplexy, 194, *note;* on dilatation and hypertrophy of left ventricle from regurgitant disease of mitral orifice, 226, *note;* on diagnosis of fatty disease of heart, 352; on seat of nervous murmurs, 512; on muscular prolongation, 525; on doubling of sounds of heart, 526, *note;* on signs from percussion in aneurism, 556.

Watson, Dr., valvular murmur overrated as an indirect sign of pericarditis by, 50.

Weakened condition of heart, 314; in pericarditis, circumstances diagnostic of, 103; in valvular disease, two kinds of signs of, 175; causes of, 314; in pericarditis, 315; novel character of impulse in a case of, 344; case of, 554, *note;* sudden occurrence of, in fever, 458; angina most commonly occurs in, 501; muscular prolongation and vermicular sensation connected with, 525.

Weber, Professor on use of columnæ carneæ, 261, *note.*

Weyland, remarkable case of diaphragmatic hernia by, 471.

Williams, Dr., on fatty degeneration of heart, 335; on arcus senilis in the same, 354.

Wine, use of, in fever, 103, *note;* importance of state of heart in reference to, 391, 399, 406, 454, 456, 459; effects of, in reducing rate of pulse, 396, 407, 409; cases of free, 402, 405, 420, 425, 435; tolerance of, 455.

—— use of, in local inflammation, 103; in pericarditis, 104, 105.

Wood, Dr., on treatment of pericarditis, 100 *note.*

Wrisberg, pain in the nerves of, in cardiac disease, 508.

Y.

Young persons, palpitation of heart in, 531.

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